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SOME MOOTED POINTS IN THE PATHOLOGY OF APPENDICITIS,

WITH SPECIAL REFERENCE TO THAT FORM KNOWN AS APPENDICITIS OBLITERANS.[1]

By John B. Deaver, M.D.,
SURGEON-IN-CHIEF, GERMAN HOSPITAL, PHILADELPHIA.

Since the time it was determined that the great majority of inflammatory troubles in the right iliac fossa originated in the appendix there have been diverse opinions expressed in regard to the pathology of this affection. Especially has this been true with reference to that branch of pathology which investigates the cause of the pathological changes, viz., the etiology. The anatomical structure of the organ, together with its shape, differences of position, and its general retrograde condition, has been almost universally accepted as the chief *predisposing* cause of this inflammatory trouble. But, when we consider the *exciting* causes we find a great diversity of opinion.

Of the chief exciting factors the presence of a foreign body, such as a seed, cherry-pit, etc., was the one probably first advanced. Other writers claimed that the trouble was due entirely to micro-organisms; others, again, thought that fecal concretions played the most important part.

The idea that a foreign body must necessarily be present has given way before our more complete knowledge of the disease. The theories in regard to the micro-organismal invasion and the presence of fecal concretions have had their ups and downs, until at the present time there is no consensus of opinion in favor of either as a sole cause.

[1] Read before the College of Physicians of Philadelphia, April 7, 1897.

I propose in the paper here presented to show that both theories are tenable, in that both conditions are strong factors in the causation of appendicitis, although as a rule the formation of a fecal concretion precedes the invasion of micro-organisms. I wish to show also the relation of these causes, both of which are known by the course of the disease and by the conditions present at the time of operation to be active in the vast majority of cases.

The ideas advanced have been obtained from a study of over 1500 microscopical slides made at the German Hospital, where each case of appendicitis is systematically and thoroughly studied. A full and complete history of the patient's trouble is obtained. A description of the operation is recorded, special note being taken of the position of the appendix, the general direction which it holds, the presence or absence of pus, etc. The organ is photographed immediately after removal, and in many instances an x-ray photograph is also obtained. The appendices are then treated for microscopical work in the usual manner for section-cutting. After the specimens have been thoroughly hardened they are cut into sections of one-half centimetre each, and the diameters of the outside and of the lumen, the appearance of the different coats, and the contents of the lumen are noted. Two or more of these portions are then cut into sections, stained, and permanently mounted. The most interesting are then photomicrographed, and we are ready for a thorough study of the inflammatory trouble.

The few statistics presented have been taken from forty of the fifty cases which have been operated on at the German Hospital since January 1, 1897. These may vary somewhat from the statistics of a full year's work, but I consider them of more interest since they are the result of very recent investigations.

It is probable that every primary attack of appendicitis follows an invasion of micro-organisms. This invasion, on account of hyperæmia or deranged circulation of the organ, superinduced by kinks or twists in the mesentery of the appendix, by fatigue, traumatism, typhoid fever, influenza, etc., may take place, in a few cases, through the lymph-channels. In a vastly greater number of cases, however, it occurs through an eroded mucous membrane. The erosion is caused by masses of fecal matter, rarely by a foreign body. Fecal matter is introduced into the lumen of the appendix by contractions of the cæcum. The expulsive force of the appendix is not sufficient to expel it, and hence it remains to irritate the mucous layer, at first merely by its presence. The slight muscular movements of the appendix tend to mould the mass into a round or oblong shape. This mass is augmented by the natural secretions of the mucous membrane of the appendix and by further accessions of fecal matter from the cæcum. Gradually the concretion increases in size until it irritates by causing pressure against the

walls of the organ. This is followed by a decided erosion of the mucous membrane, and this by an invasion of the micro-organisms which are always present in fecal matter. They proliferate, and the inflammatory troubles then begin. In a few cases the increased secretion of the mucous layer may cause partial liquefaction of the fecal concretion, which may be expelled. In the majority of cases, however, the concretion remains in the lumen, where it is bound to act as an exciting cause of further inflammatory trouble.

FIG. 1.

Separation of base of appendix from its origin from cæcum, thus forcing the fecal contents of the latter into the peritoneal cavity.

Following the invasion of the micro-organisms we find the general conditions common to any inflammation of the mucous membrane. It must be borne in mind that this invasion takes place only at the site of the dissolution of the mucous membrane, although the micro-organisms are liable to spread rapidly through all the walls of the organ. With the exception of the point of invasion the mucous membrane may be, and often is, absolutely normal and healthy; at other times it shows the effect of inflammation which has extended by continuity of structure.

Almost immediately after the invasion there is an attempt at reconstruction on the part of nature. A round-celled infiltration takes place, with at times a deposition of fibrous connective tissue. Unless interrupted in its course by nature or the surgeon, the process will continue not only through the walls of the organ, but to the surrounding structures, giving rise to that stage of the appendiceal inflammation which is known as para-appendicitis. Here we have to deal with troubles which are known to all operators who have seen many cases that were allowed to take their course. In the first place, there is a bank of lymph thrown out for the purpose of protecting the general peritoneal cavity from infection. In case of an abscess-formation this wall is of the greatest importance in saving the patient from a purulent peritonitis. That an abscess may cause much trouble *per se* if allowed to remain long in the right iliac fossa is beyond dispute. I have seen several cases where the pus burrowed toward the liver, under the limiting membrane, along the iliac vessels into the thigh, etc. This causes at times a condition beyond our power to control. I remember one case in which there was present a large quantity of foul pus which had burrowed under the limiting wall, and formed pockets which could be found only by the most thorough search. Time and again I thought that I had the cavity perfectly clean, but would be confronted by another welling up of the purulent matter from some unknown site. After general septicæmia had ended the patient's life, the post-mortem revealed many other pockets, which had not been reached, hidden beyond the limiting membrane.

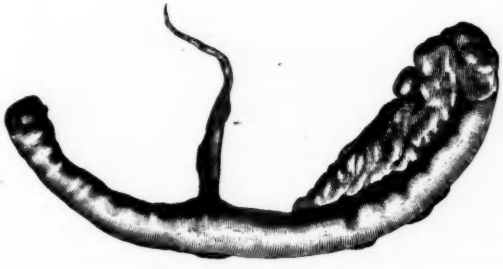
The relation of the appendix to this limiting membrane plays a most important part in the prognosis of appendicitis, and on one point I wish to lay special stress. *In a vast majority of cases the limiting membrane does not include or surround the entire organ.* The base, as a rule, is the part left exposed, and I have seen some few cases where a perforation had taken place through this portion of the appendix, with a resulting general purulent peritonitis. The abscess-cavity at the same time remained intact, and but little trouble was found in dealing with it. Since the base of the organ is without the limiting membrane it does not enter into the formation of the wall, nor is it protected by the wall, and, therefore, any pathological change which may take place in that part will necessarily affect the peritoneum in contact with it, and, naturally, the general peritoneal cavity. In most cases this exposed condition of the base does not lead to any untoward effects, but there are other cases in which perforation has taken place with fatal results. (Fig. 1.) Why nature allows this condition of affairs to exist no one can say, but that it does exist cannot be denied. The body of the appendix is generally a constituent part of the limiting membrane, and is thus surrounded by a mass of exudate which prevents it from perforating or causing much trouble. The tip may be within the wall—that is, in the

abscess-cavity; or it may be a part of the wall or without the wall—that is, in the general peritoneal cavity. In the latter instance perforation may take place with results similar to those found in perforation at the base—that is, general purulent peritonitis. Perforation does take place from the extreme tip of the organ at times, as is well shown in Fig. 2; but it is generally in those cases which have gone on to a chronic condition, with occlusion of a portion of the lumen of the appendix. These cases I will discuss later.

FIG. 2.

In those cases presenting no pus-formation, but appendiceal peritonitis, we will find adhesions from any portion of the length of the appendix to some of the surrounding structures or to other portions of the organ. These, if allowed to remain in place, play an important part in causing kinks, twists, etc., in the appendix. It is generally known that the adhesions are always more numerous and of greater

FIG. 3.

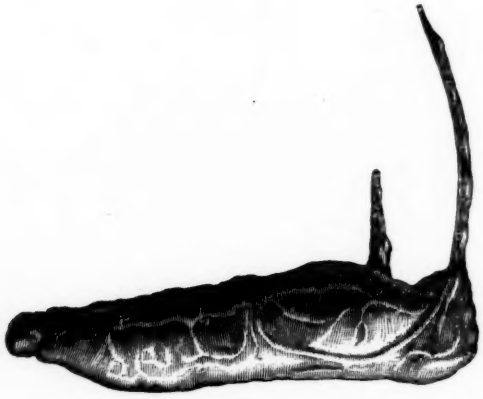


pathological significance in those cases that have been repeatedly visited by the dread inflammation. Some of the adhesions that were formed during the inflammatory condition of the organ are well shown in Figs. 3 and 4. These might well be considered as a fruitful factor in the form of ileus which is caused by a band.

In those cases also where we do not find pus-formation we may meet with one or more of the several following conditions: the condition of the lumen after an attack of appendicitis is such that it is an almost constant cause of further attacks. After the round-celled infiltration

has taken place we find contraction of the tissue which nature has thrown out to repair the damage. This will cause a constriction of the lumen at that point, with, generally, occlusion of the entire calibre. Out of the forty cases that have been operated on at the German Hospital since the first of the year, twenty-five of them were partially occluded—that is, there was a complete obliteration at one or more points in the length of the lumen. This fact alone accounts for many secondary attacks of inflammation and of much suffering in some cases where there is no succeeding inflammatory trouble. As stated, we find the mucous membrane healthy in many parts of the lumen. Should this be the condition of the membrane to the distal side of the obstruction in the lumen, we shall naturally have the secretions and débris thrown out by the mucous layer confined. As the amount increases nature endeavors

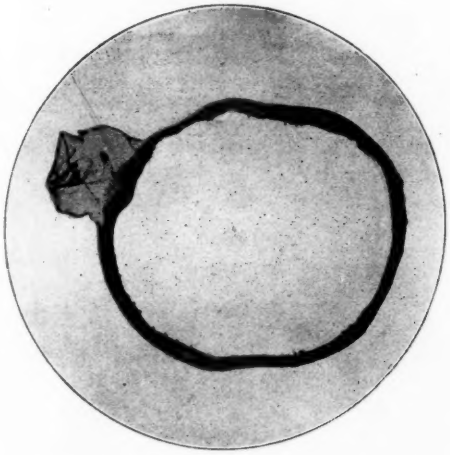
FIG. 4.



to expel it. This can be accomplished only by forcing the accumulated material through the obstruction or through the walls of the organ, which, by ulceration, have been weakened and distended. If through the obstruction, there is a tearing asunder of the weakest portion of the constriction—that is, the walls of the appendix are forced apart and the lumen reopened by breaking down of the round-celled infiltration which has completed the occlusion at that point. This is the cause of much suffering on the part of the patient, and accounts for many attacks of severe, excruciating pain with which the possessor of a chronic appendicitis is very often familiar. The pain is caused not by inflammation, to which it has been generally ascribed, but by the pressure on the delicate nerve-endings with which the appendix is well supplied. In these cases there is absolutely no inflammation present at the time of the pain.

This expulsion of contained material may take place time and again, until the barrier has become strong enough to withstand all efforts nature may make to force it open. We then find a condition which will be followed by ballooning of the distal portion of the organ, forming what has been termed a cystic appendix. As long as the walls are strong enough to stand the pressure we will have but few symptoms, except the pain caused by pressure on the nerve-endings. The walls finally become so thin that they rupture, and we then have a peritonitis, abscess, or what not, to deal with. Fig. 5 well shows this condition of ballooning. The illustration is a photomicrograph of a section. While these photomicrographs may not show clearly all the microscopical changes in the organ as a result of inflammation, yet they will answer the purpose of illustration better than any other means I know.

FIG. 5.



Of more significance than the secretions and débris of the mucous membrane are the fecal concretions that are so often walled off in the lumen of the appendix. Of the forty cases, twenty contained fecal concretions. Some of these were confined to the distal side of an obstruction, and, if allowed to take their course, could give but one result—rupture of the organ, with expulsion of the fecal matter into the abdominal cavity. The concretion being too large and too consistent to be forced through the obstruction, must be liberated through the walls of the organ unless the latter be removed from the body. Figs. 6 and 7 show a concretion in section. Of the forty cases seven had ruptured. Foreign bodies sometimes play a *rôle* in this connection, as Fig. 8 clearly shows. The

illustration is from a blue-print of an x-ray photograph. Near the tip of the organ will be seen a dark spot, which investigation proved to

FIG. 6.

FIG. 7.

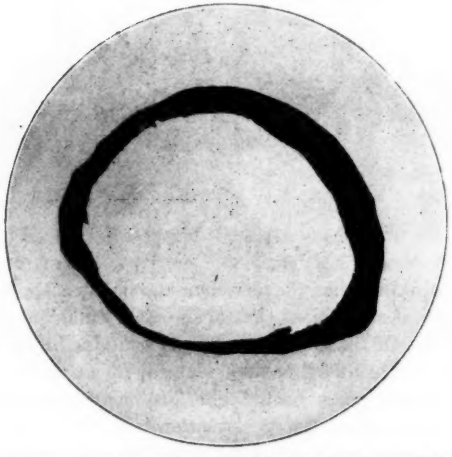


Fig. 7 is a photomicrograph of a section from which the fecal concretion has been removed. It shows dilatation of the lumen and erosion of the mucous coats.

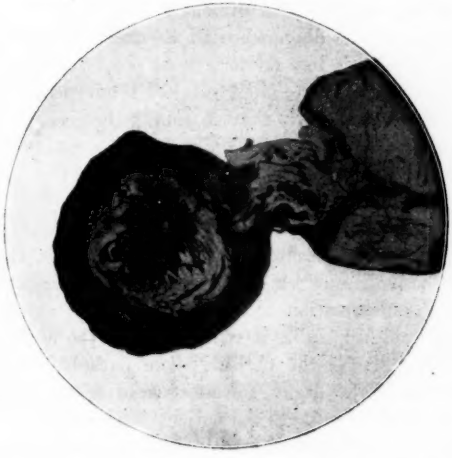
have been caused by a small particle of lead. How it got there is a nice question to decide. It was probably a shot taken into the alimen-

tary tract while the patient was eating game. The illustration does not show it clearly, but just to the proximal side of the piece of lead was a constriction. But one result could have occurred in this instance had the appendix been allowed to remain in the abdominal cavity, viz., rupture at or near the extreme tip.

FIG. 8.

Partial occlusions are often formed in different portions of the same lumen, and several may be found at different points along its length. There is always increased suffering in these cases on account of nature's efforts to expel any contained material. When these occlusions are so

FIG. 9.



numerous that they gradually work together and close the entire lumen we have a condition that has been described by many writers as *appendicitis obliterans*. Examination of appendices removed shows that some of these cases are the result of occlusion that begins at the tip and gradually works its way to the base, pushing before it any material that may

be found in the lumen. Although the result of this process has been termed *appendicitis obliterans*, that term is a misnomer. There is no obliteration of the appendix excepting that small amount which is brought about by contraction and atrophy of the unused organ. What does take place is entire obliteration of the lumen of the organ, but not of the organ itself. But one of the fifty cases operated on this year shows this condition, although I had several of them prior to January 1st. The history of one case from last year may be of interest. Dr. B., aged forty years, had had one attack of acute appendicitis, from which he apparently recovered with little trouble. He noticed, however, that the pain persisted even after he was able to leave his room and attend to his practice. The pain was paroxysmal, excruciating at times, often remittent, but never entirely absent. The appendix was removed; the patient made a good recovery, and has been free from pain since. The pathological examination of the organ showed that the lumen was entirely occluded. There was no evidence of active inflammation. The site of the lumen was replaced by a mass of round-celled infiltration and fibrous connective tissue. Fig. 9 is a photomicrograph of one of the sections, and is a splendid illustration of the condition of the entire lumen. I believe that the pain from which this patient suffered was due to impingement of the small nerve-endings in the appendix, and not to any inflammatory trouble present at the time. I am not sure that these cases can be diagnosticated as such before the operation, although I think there is less tenderness on pressure; that there must be more decided pressure to elicit marked tenderness; and that the patient suffers more constantly than is usually the case.

There is but one other point to which I wish to call your attention, and that is the presence of a circumscribed collection of pus in the walls of the appendix. Three of the forty cases studied showed this condition. It is scarcely necessary to state that recovery from such a condition could be accomplished only by the removal of the organ.

A study of these changes which are wrought in an appendix that has been the seat of inflammation convinces me more decidedly than ever that the sooner such appendix is removed from the abdominal cavity the better it will be for the patient. The pathology is decidedly in favor of removal of the organ, for most cases show a condition that must result in serious trouble to the patient if operation be declined.

I would like to add that no two appendices show exactly the same pathological changes, and that we but seldom find the many sections of any given organ agreeing exactly with each other.

To summarize the examinations of the forty cases, I would state that twenty of them contained a fecal concretion; twenty-five were partially occluded—that is, had an incomplete obstruction at one or more points in the length of the lumen; one was entirely occluded—that is, took that

form generally described as appendicitis obliterans; seven were ruptured; three contained abscess in the walls; one contained a foreign body (a piece of lead).

Serum Diagnosis. The medical profession has been startled with a new blood diagnosis for enteric fever, and the principles laid down by these discoveries will doubtless lead to wider observations for diagnostic purposes. The experiments of Durham, of London, "On Special Action of Serum on Immunized Animals" (read before the Royal Medical Society of London, January, 1896), show conclusively that special micro-organisms, such as cholera vibrios, colon-bacilli, and typhoid bacilli, produce special forms of immunity in animals by injection of living or dead cultures. Such immune serum, when mixed with pure cultures of the same micro-organism as was used to produce immunity, will show the following reaction: the micro-organisms will lose their power of motility, if motile, and produce clump-formations. Durham's experiments showed that serum immunized by colon-bacilli, when inoculated by active culture of the same species, exhibits reactions of stoppage of motility, clump-formation, and the serum remains clear after twenty-four hours' incubation; control-tests made with normal serum showed no loss of motility, no clump-formation, and the serum became uniformly cloudy. The colon-bacillus was also tested with serum immunized by typhoid and cholera, but showed no loss of motility, no clump-formation, and the serum became uniformly cloudy.

The infections of appendicitis are in a very large proportion due to the varieties of colon-bacilli, sometimes mixed with staphylococcus, streptococcus, and bacillus pyocyaneus. With these facts in mind we have taken the blood from three cases of acute appendicitis with abscess-formation, and have tested the same by the method employed by Johnson in the Widal test. Control-tests were also made with normal blood. It was noted that in each the loss of motility was apparent and that there was slight clump-formation. The cases when operated on showed abscess-formation containing the colon-bacillus in almost pure culture.

Observations in this line are too recent to warrant lengthy records, but I wish to inform the Fellows of the College that special study of the infections in appendicitis is being made, the results of which will be made public. Serum diagnosis may be of value in differentiating typhoid fever from appendicitis, vague abscess-formation, symptoms produced by severe catarrhal conditions, and appendicitis obliterans.

I recently operated upon a child, thirteen years old, in whom the diagnosis of pus was made chiefly from the great degree of tenderness present, and in whom this test had been made, but of which I was not aware. The operation revealed pus. The test showed the colon-bacillus immobile. While I believe this test is a valuable one, yet I would not

be willing, in the majority of cases, to defer operation until the test be made if it would entail much delay, believing, as I do, that delay is too dangerous.

In conclusion, I wish to tender my thanks and credit to the resident staff of the German Hospital, particularly Mr. M. J. Wilbert, Drs. C. Freese, Kalteyer, and Blair, for the work which has rendered this paper possible.

CLASSIFICATION OF ACUTE PERITONITIS.[1]

By N. Senn, M.D., Ph.D., LL.D.,
PROFESSOR OF PRACTICE OF SURGERY AND CLINICAL SURGERY, RUSH MEDICAL COLLEGE; ATTENDING SURGEON, PRESBYTERIAN HOSPITAL; SURGEON-IN-CHIEF, ST. JOSEPH'S HOSPITAL.

An intelligent and systematic discussion of acute peritonitis must be necessarily based on a rational classification. A great deal that has been said and written on this subject from the distant past until the present time is worthless from a scientific as well as a practical standpoint, owing to a lack of a proper classification. The ordinary terms used to designate the different forms of peritonitis are differently interpreted and applied by pathologists and clinicians. Acute inflammation of the peritoneum is produced by so many different causes and assumes such varied clinical aspects that it is extremely difficult to formulate a satisfactory classification. A discussion of the etiology, differential diagnosis, prognosis, and treatment of acute peritonitis except upon the basis of a clear and comprehensive classification is fruitless, misleading, and usually results in the deduction of erroneous and often dangerous conclusions. The classification should include the anatomy, pathology, and etiology of the disease to be of value in rendering a correct diagnosis, a reliable prognosis, and in enabling the physician and surgeon to advise and apply effective therapeutic measures.

1. Anatomical Classification. An accurate anatomical diagnosis is necessary for the purpose of locating the inflammatory process correctly or to trace the connection between it and the organ primarily the seat of infection. During the beginning of the attack and in cases of localized peritonitis the inflammation can usually be located without much difficulty, while the reverse is often the case after the disease has become diffuse. The inflammation may commence and spread from either surface of the serous membrane, visceral or parietal.

(*a*) *Ectoperitonitis.* An inflammation of the attached side of the peritoneum is called ectoperitonitis. As compared with inflammation of the serous surface, this inflammation of the subendothelial vascular con-

[1] Read before the Congress of American Physicians and Surgeons, May 5, 1897.

nective tissue is characterized clinically and pathologically by intrinsic tendencies to limitation of the inflammatory process. The mechanical and anatomical conditions for diffusion of the infection are less favorable than when the free surface of the membrane is affected. Ectoperitonitis, however, in certain localities may become quite diffuse when the cavum Retzii (Wm. Gruber) or the retroperitoneal space on either side of the spinal column is the seat of a suppurative inflammation. In the latter locality a paranephric or spondylitic abscess is often the cause of an extended ectoperitonitis, the extent of the disease corresponding with the size of the subperitoneal abscess. In infected wounds of any part of the abdominal wall in which the peritoneum is exposed, but not perforated, the primary ectoperitonitis is occasionally followed by the extension of the infection to the serous surface through the lymphatics, or the direct extension of the infective process through the tissues until it reaches the endothelial lining. Peritonitis of a visceral origin is always preceded by ectoperitonitis, whether the infection reaches the peritoneal cavity through a perforation or by progressive extension of the infection from the primary focus through the tissues until it reaches the free peritoneal surface.

(*b*) *Endoperitonitis.* What is usually spoken of and described as peritonitis is an inflammation of the serous surface of the peritoneum, which, anatomically speaking, is an endoperitonitis. Endoperitonitis not infrequently leads to ectoperitonitis and the formation of subserous abscesses. In inflammation of the serous coat of the intestine the peritoneum is always loosened and frequently extensively detached by the secondary ectoperitonitis.

(*c*) *Parietal peritonitis.* Inflammation of the serous lining of the peritoneal cavity is called parietal peritonitis. It may occur as a primary affection in penetrating wounds of the abdomen, but more frequently it is met with as a secondary disease in consequence of the extension of an infection from one of the abdominal or pelvic viscera, or perforation into the peritoneal cavity of a visceral ulcer or a subserous or visceral abscess. Visceral peritonitis is always associated with parietal peritonitis, and parietal peritonitis is only absent in visceral peritonitis when the inflammation remains limited and the parietal peritoneum is protected against infection by plastic exudations. In the female, parietal peritonitis of the pelvic floor usually follows in the course of an extension of an infective process from the internal genital organs through the lymphatics, rupture of a visceral or connective-tissue abscess into the peritoneal cavity, or leakage of septic material from the Fallopian tubes.

(*d*) *Visceral peritonitis.* Inflammation of the peritoneal investment of any of the abdominal or pelvic organs is known as visceral peritonitis. The inflammatory process is seldom limited to a single organ, as during the course of the disease adjacent organs or the parietal peritoneum will

surely become involved. In diffuse peritonitis the whole peritoneal sac and the serous covering of all of the abdominal organs are affected. The nomenclature of visceral peritonitis is a lengthy one, as it includes all of the abdominal and pelvic organs which, when the seat of a suppurative inflammation, may become the primary starting-point of an attack of localized or diffuse peritonitis. The mesentery and omentum are modified anatomical forms of the peritoneum, and when the seat of inflammation we speak of mesenteritis and epiploitis. Peritonitis involving the serous covering of any abdominal organ, and arising in consequence of an inflammation of the organ which it invests, is designated by the prefix *peri*, and the noun used to indicate the organ primarily affected in a state of inflammation which has given rise to the following terms: peri-gastritis, peri-enteritis, peri-typhlitis, peri-appendicitis, peri-colitis, peri-hepatitis, peri-splenitis, peri-cystitis, peri-salpingitis, and peri-oöphoritis.

(*e*) *Pelvic peritonitis.* Inflammation limited to the peritoneal lining of the pelvis and its contents is known clinically and anatomically as pelvic peritonitis. It is an affection almost entirely limited to the female sex, and in the majority of cases is caused by extension of gonorrhœal infection from the Fallopian tubes, or a mild form of pyogenic infection from the uterus, its adnexa, or connective tissue of the parametrium.

(*f*) *Diaphragmatic peritonitis.* Inflammation of the under surface of the diaphragm is described as diaphragmatic peritonitis, and when it assumes a suppurative type and remains limited leads to the formation of subdiaphragmatic abscess. This acute localized form of peritonitis is usually secondary to suppurative affections of the liver and gall-bladder and perforating ulcers of the stomach and duodenum.

2. Etiological Classification. The classification of peritonitis upon an etiological basis is of the greatest importance and practical value. The nature of the exciting cause frequently determines the anatomical and pathological varieties. It likewise has a strong bearing upon the prognosis, and often furnishes positive indications as to the methods of treatment that should be adopted. Peritonitis, like every other inflammatory affection, is always the result of infection with pathogenic microbes usually of the pyogenic variety. The etiology must consider the different avenues through which the microbes find their way into the peritoneal cavity.

(*a*) *Traumatic peritonitis.* Primary peritonitis has usually a traumatic origin—that is, the injury establishes between the peritoneal cavity and the surface of the body, or some of the hollow abdominal or pelvic organs, a communication through which pyogenic bacteria enter in sufficient quantity and adequate virulence to cause an acute inflammation. Traumatic peritonitis is most frequently caused by penetrating-wounds of the abdominal wall, the uterus, and lower portion of the œsophagus.

In some cases the penetrating wounds of the chest extend into the abdominal cavity through the diaphragm. Contusions and lacerations of the abdominal organs often cause peritonitis by extravasation of the secretions or excretions of the injured organ. Not infrequently the injury is followed by a circumscribed suppurative inflammation in the injured organ, which later is followed by diffuse peritonitis from perforation of an abscess or the injured wall of any part of the gastro-intestinal canal, in which case the peritonitis follows as a secondary affection.

(*b*) *Idiopathic peritonitis.* The occurrence of peritonitis without an antecedent injury or suppurative lesion is doubted by many. It is an exceedingly rare affection, since pathologists and surgeons have brought by their investigations and observations the peritoneal inflammation with few exceptions in connection with a neighboring or distant primary suppurative lesion. It is certainly much more rare than primary inflammation of the pleura and pericardium. It is too early to deny *in toto* the existence of so-called idiopathic peritonitis, but future bacteriological examinations of the inflammatory product will no doubt reveal a microbic cause in all such cases. Leyden found in a case of primary peritonitis in the inflammatory exudate diplo- and streptococci. As an isolated affection peritonitis is found most frequently in females during or soon after menstruation; it is probable that the pyogenic bacteria multiply in the blood which accumulates in the uterus and reach the peritoneal cavity through the Fallopian tubes. It is said to have occurred in consequence of exposure to cold, and is then known as rheumatic peritonitis. Occasionally it has been observed as one of the remote manifestations of Bright's disease, pyæmia, and the acute eruptive fevers.

(*c*) *Perforative peritonitis.* Perforation of an ulcer of any part of the gastro-intestinal canal, or of an abscess of any of the abdominal or pelvic organs, or of the abdominal wall into the peritoneal cavity, is by far the most frequent cause of acute peritonitis. Two important and frequent causes are appendicitis and suppurative salpingitis. If localized inflammation develops over the ulcer or abscess before perforation takes place, the general peritoneal cavity is often protected by firm adhesions before the accident occurs, and the peritonitis remains circumscribed. If, however, the contents of the gastro-intestinal canal or the abscess-cavity reach the free peritoneal cavity, a diffuse septic peritonitis sets in, which usually destroys life within from twelve to seventy-two hours unless prompt surgical treatment is resorted to. Experimental research as well as clinical observations has demonstrated that the intestinal wall, when paretic or gangrenous, becomes permeable to the microbes contained in the intestinal canal. In many cases of intestinal obstruction, acute and chronic, death results from septic peritonitis after the intestine has become paretic or gangrenous.

(*d*) *Metastatic peritonitis.* This form of peritonitis occurs like other

metastatic affections in connection with a suppurative or infectious process disconnected from the peritoneum. It is more rare than metastatic pleuritis, and is seldom seen except as a pyæmic lesion. In very rare cases it develops in the course of many of the acute infectious diseases, as scarlatina, smallpox, erysipelas, rubeola, and even varicella. It also occurs frequently in the course of septicæmia and pyæmia. Andral and Desplatz have seen it occur during attacks of acute articular rheumatism. It has also been observed in scorbutic subjects and in patients suffering from valvular disease of the heart.

(*e*) *Puerperal peritonitis.* Peritonitis occurring in connection with septic diseases of the puerperal uterus has for a long time been known as puerperal peritonitis. The infection may extend from the endometrium through the Fallopian tubes, or it follows the lymph-channels, or the thrombosed infected uterine veins. Infection through the lymphatics usually results in rapidly fatal diffuse septic peritonitis, while in thrombophlebitis there is a greater tendency to localization unless the thrombi disintegrate and cause embolism and pyæmia.

(*f*) *Peritonitis infantum.* Peritonitis attacks most frequently children, young adults, and women during the childbearing period of life, but no age is exempt.

Fœtal and intrauterine peritonitis. If the disease attacks, as it occasionally does, the fœtus in utero, it often results in death before or soon after the birth of the child; most frequently death ensues during the seventh to the ninth month of gestation. Prenatal peritonitis is frequently associated with syphilis. In most cases the disease is only detected at the post-mortem; in others death, if the child is born alive, is preceded by meteorismus, icterus, and œdema of the legs.

Peritonitis neonatorum. Infection takes place during the first few weeks after birth through the imperfectly healed ulcerating umbilicus. It has been met with most frequently in children whose mothers were afflicted with puerperal fever. Besides peritonitis, symptoms of pyæmia appear.

3. Pathological Classification. The pathological conditions which characterize the different varieties of peritonitis must necessarily be considered in classifying this disease. The pathological classification is based almost entirely upon the gross and microscopical appearances of the inflammatory exudation and transudation.

(*a*) *Diffuse septic peritonitis.* Every acute peritonitis is septic in so far that phlogistic substances reach the general circulation from the inflammatory lesion, and in that frequently the inflammation terminates in suppuration; but the term septic peritonitis should be limited to those cases of diffuse septic peritonitis in which, as a rule, death occurs in a few days, and before any gross pathological conditions have had time to form. It is a disease that is almost uniformly fatal with or without

operative treatment, the patients dying from the effects of progressive sepsis. The claim of operators to have cured such cases by laparotomy must be accepted with a good deal of allowance. The microbes which produce this form of peritonitis are those which follow the lymph-spaces, and are rapidly diffused not only over the entire peritoneal surface, parietal and visceral, but also through the subserous lymphatic channels. The disease is observed most frequently after perforation into the free peritoneal cavity of an abscess containing septic pus, rupture or perforation of any of the abdominal or pelvic viscera containing septic material, gunshot- or stab-wounds of the abdomen with visceral injury of the gastro-intestinal canal, and occasionally as the result of infection during laparotomy. The gravest form of puerperal fever is a diffuse septic peritonitis. The subjects of this variety of peritonitis die so soon after the beginning of the disease that at the post-mortem, or, if the abdomen is opened during life, at the operation, no gross tissue-changes are discovered. Besides a slightly increased vascularity, nothing is found to indicate the existence of peritonitis. The septic material formed in large quantities and of great virulence is rapidly absorbed by the stomata of the under surface of the diaphragm discovered and described by von Recklinghausen.

(*b*) *Putrid peritonitis.* The inflammatory product in this form of peritonitis is a scanty brown or reddish brown fetid fluid. It occurs most frequently in connection with grave forms of puerperal metritis. It is usually associated with more or less gangrene or ulceration of the organ or parts primarily affected, as uterus, intestine, or abdominal wall.

(*c*) *Hemorrhagic peritonitis.* The ascites which so frequently develops in consequence of malignant or tubercular disease of the peritoneum is composed of serum frequently stained with blood, but in acute peritonitis the transudate is occasionally hemorrhagic by the tearing of vascular adhesions, and rhexis is also observed in persons suffering from peritonitis who are greatly debilitated, scorbutic, or intemperate, and occasionally when peritonitis occurs in patients suffering from typhoid fever. It is met with most frequently in the pelvis upon the posterior surface of the uterus and vagina, and in men behind the bladder. The inflammatory product appears in the form of brown patches which are composed of delicate and very vascular villi. These villi, from their unfavorable location, are subjected to frequent mechanical disturbances, and when injured bleed, giving rise to the so-called retrouterine or retrovesical hæmatocele.

(*d*) *Suppurative peritonitis.* Suppurative peritonitis — that is, an inflammation of the peritoneum which results in the formation of pus — is always more or less circumscribed. This form of peritonitis is the most frequent, and is generally associated with more or less fibrinoplastic exudation. The pus is either serous, seropurulent, or may reach the

consistence of cream, when it is usually of a yellow color. The accumulation of pus may be so large that upon opening the abdominal cavity it may appear as though the entire peritoneal cavity and all of the organs contained within were implicated; but a careful examination will almost always reveal the fact that a large part of the peritoneal cavity and many of the organs were shut out from the inflammatory process by plastic adhesions. Suppurative peritonitis must, therefore, be regarded from a practical standpoint as a circumscribed inflammation. The appearance and character of the pus are often greatly modified by the admixture of an extravasation accompanying the perforative lesion which produced the peritonitis. If the pus is thin, serous, we speak of a *seropurulent peritonitis*. It is a serous peritonitis with the formation of pus in sufficient quantity to render the serum more or less turbid. This subvariety of suppurative peritonitis is, without exception, in combination with fibrinous exudations which tend to limit the extension of the infective process. Sedimentation of the solid constituents takes place, so that the fluid contains more of the solid constituents in the most dependent portion of the affected district.

(*e*) *Serous peritonitis.* Independently of malignant and tubercular disease of the peritoneum, circumscribed hydrops of the peritoneal cavity is caused by a very mild form of peritonitis, the pus-microbes present not being sufficient in quantity to produce pus. Patients usually recover rapidly from this form of peritonitis. The slight alterations of the peritoneum produced by the inflammatory process do not interfere with the transudation of serum, and resorption is effected as soon as the inflammation subsides and the normal absorptive function of the peritoneum is restored. Serous peritonitis is usually more or less complicated by fibrinous peritonitis, as fragments of fibrin are often found suspended in the fluid. The serum is generally somewhat turbid, not transparent, grayish-yellow or reddish in color. As long as the fluid is limited in quantity it gravitates toward the most dependent parts of the abdominal cavity, in the small pelvis; when more copious it reaches the upper portions of the peritoneal cavity, and seeks first the depression on each side of the spinal column.

(*f*) *Fibrinoplastic peritonitis.* Peritonitis in which plastic exudations are formed and pus is absent or scanty is called fibrinoplastic peritonitis. *Exudative peritonitis* and *peritonitis adhesiva seu sicca* have been used as synonymous terms for this variety of peritonitis. It is usually a secondary process following a primary affection of one of the abdominal or pelvic organs, and denotes a mild form of infection, the extension of which becomes limited by firm adhesions. The inflammation results in a plastic exudation with little or no effusion. The character of the exudate depends on the intensity and quality of the bacterial cause. The exudation is often so copious that it has been mistaken for malig-

nant disease. Goldberg reports two such cases. The symptoms were marked cachexia, ascites, uncontrollable diarrhœa, and apparent tumor deep in the abdomen. The distinguishing features of this form of peritonitis from abdominal tumor are the less circumscribed outline, the less resistance offered, the more regular surface, and the fact that the ascitic fluid is not bloody, but serous or seropurulent. The exudation in the course of time contracts and results in strong bands of adhesion which frequently flex and distort the organs to which they are attached, which has given rise to another term—*peritonitis deformans.*

4. Bacteriological Classification. As the essential cause of peritonitis is always the presence and action of pathogenic microbes and their toxins upon the peritoneum, and as the character of the inflammatory process is largely influenced by the kind of microbes which produced the infection, a bacteriological classification is of the greatest scientific and practical importance. All pus-microbes present in sufficient quantity and virulence in the peritoneal cavity can produce peritonitis. Experiments as well as clinical observation have shown, however, that their action is enhanced by local conditions which favor their growth and reproduction. Injuries or antecedent lesions of the peritoneum and the presence of putrescible substances furnish such predisposing and exciting conditions.

(*a*) *Streptococcus infection.* The streptococcus pyogenes is the microbe which is most frequently found in the tissues in cases of septic peritonitis. The infection spreads so rapidly over the peritoneal surface and through the subserous lymphatics that death, as a rule, occurs from septic intoxication before a sufficient length of time has elapsed for any gross pathological conditions to form. Absence of fibrinous exudate and effusion is the most striking negative finding at operations and necropsies. Streptococcus infection is the immediate cause of the most fatal form of puerperal peritonitis. Fränkel has found the streptococcus pyogenes in a great variety of puerperal diseases, especially in cases in which the local affection implicated the lymphatic vessels. In such cases the microbes found entrance into the pelvic tissues from abrasions of the vagina or uterus, and by extension of the inflammatory process the broad ligaments and the peritoneum are successively reached. After the peritoneum has once been infected, rapid diffusion takes place, and finally the diaphragm and pleuræ are implicated in the same process, and the patient dies from the effects of progressive sepsis.

(*b*) *Staphylococcus infection.* In peritonitis caused by staphylococcus infection the intrinsic tendency to localization of the disease is more marked, the inflammation results more often in circumscribed suppuration and limitation of the infective process by copious fibrinoplastic exudations. As a rule, the inflammation terminates in the formation of

thick, cream-colored pus. Different forms of staphylococci are often seen in the same inflammatory product.

(*c*) *Pneumococcus infection.* It is now well known that pneumonia is produced by different microbes, but the diplococcus is found in about 80 per cent. of all cases. It is this microbe which occasionally is found as the bacteriological cause of acute suppurative peritonitis. Weichselbaum has found the diplococcus of pneumonia unaccompanied by any other micro-organism in three cases of peritonitis. In one case the peritonitis and acute pneumonia occurred simultaneously; in the other, double pleuritis followed the peritonitis; but in the last case the peritonitis was undoubtedly primary, and, in the absence of any other microbes in the inflammatory product, must have been caused solely by the diplococcus of pneumonia.

Etheridge has described three cases of abscess of the ovary complicated by plastic peritonitis, in the contents of which the diplococcus alone was found. Le Gendre reports a case of peritonitis in a girl of eighteen years; the pus was yellowish-green, lumpy, and of a fibrinous consistence, and contained a pure culture of the pneumococcus. The author found altogether eleven cases recorded, eight of which had been fatal. Another case is reported by Veillon. The suppuration caused by pneumococcus infection is almost invariably attended by copious fibrinoplastic exudation.

(*d*) *Bacillus coli commune infection.* The bacillus coli commune, a microbe that constantly infests the intestinal canal, is in a fair percentage of cases the bacteriological cause of acute peritonitis. This microbe possesses pyogenic properties, and in intestinal paresis and perforations escapes into the peritoneal cavity and produces usually a pathologically mixed form of peritonitis—that is, suppurative and fibrinoplastic peritonitis. In thirty-one cases of peritonitis examined by Fränkel this microbe was found in nine. In eleven cases, seven gave mixed cultures, and in three of these the colon-bacillus predominated. The same author has shown that pure cultures injected into the abdominal cavity of rabbits cause typical peritonitis.

(*e*) *Gonococcus infection.* In the peritoneal cavity the gonococcus produces a plastic peritonitis, and sometimes localized suppuration. Salpingo-peritonitis and more diffuse pelvic peritonitis are most frequently caused by gonococcus infection. "The proper character and results of the pathogenous activity of the gonorrhœic microbes are, therefore, seen pure and unadulterated in the tubes. They cause purulent inflammation of the mucous membrane, but the surrounding connective tissue remains free from them. The gonorrhœic tubal pus is evacuated into the peritoneum; and, whereas in other conditions the bursting of an abscess into the abdominal cavity is followed by the gravest consequences, in this case the whole process terminates with a circumscribed inflammation

encapsulating the exuded pus. The cause of this difference is the varying pathogenic value of the organisms which are contained in the pus. A puerperal pelvic cellulitic abscess bursting into the peritoneum causes general peritonitis, because it contains pyogenic streptococci, which rapidly multiply in serous cavities and are capable of exerting the most deleterious effects. Gonorrhœal tubal pus cannot do this; its microbes do not find in the peritoneum conditions for their increase; the pus, therefore, acts as an aseptic foreign body, becomes encapsulated, and is finally absorbed" (Sinclair).

(*f*) *Tubercular infection* The rapid diffusion of the tubercle-bacillus in the peritoneal cavity, either through the circulation or by rupture of a tubercular abscess into the peritoneal cavity, or by extension from a tubercular salpingitis, occasionally gives rise to a form of acute peritonitis characterized as such in a modified way by the clinical manifestations which accompany it. According to the intensity of the infection or the degree of susceptibility of the patient to the action of the tubercle-bacillus, the disease assumes one of the following pathological forms: 1. Tubercular ascites. 2. Fibrinoplastic peritonitis. 3. Adhesive peritonitis. Suppuration only takes place when the tubercular product becomes the seat of a secondary mixed infection with pus-microbes.

5. Clinical Classification. From a practical standpoint the clinical classification is the most important. A modern clinical classification must be based on the location, causes, and pathological types of the inflammatory process as outlined above. Upon a correct clinical differentiation between the various forms of peritonitis as seen at the bedside depends largely the adoption of a rational course of treatment. The recognition of the disease no longer completes the diagnosis for the physician and much less for the surgeon. A diagnosis for the careful physician and conscientious surgeon must include the location, extent, causation, and pathology of the disease. From the information gained from the classification already made must be obtained the material upon which to base a clinical classification. Such classification should serve as a guide in differentiating between the cases which demand surgical intervention and the cases which can be trusted to medical treatment.

(*a*) *Ectoperitonitis.* Abscess-formation in the subperitoneal connective tissue, as seen most frequently in the pelvis in women, in the cavity of Retzius in men, and in the retroperitoneal space in both sexes, is always attended by inflammation of the outer surface of the peritoneum, and is not infrequently followed by extension of the infection through the lymphatic spaces to the free surface, and exposes the patients to the risks of perforation of the abscess into the free peritoneal cavity, septic diffuse peritonitis, and death. Such abscesses should be recognized and accurately located sufficiently early to prevent such serious complica-

tions by an extraperitoneal incision and drainage; or, if the abscess is of tubercular nature, by tapping, evacuation, and iodoformization.

(*b*) *Diffuse septic peritonitis.* This form of peritonitis is characterized clinically by the gravity of the general symptoms from the very incipiency of the disease; pathologically, by the rapid diffusion of the infection over the entire serous surfaces, visceral and parietal; bacteriologically, by the presence in most of the cases of the streptococcus pyogenes in the inflamed tissues. Staphylococci, pneumococci, and the colon-bacillus may also be the cause of rapidly spreading diffuse peritonitis. This form of peritonitis usually follows penetrating-wounds of the abdominal cavity complicated by visceral injuries of the gastrointestinal canal, contusion or laceration of any of the abdominal or pelvic organs, in rupture of an abscess or ulcer into the free peritoneal cavity, or the extension of a septic lymphangitis from any of the abdominal or pelvic organs to the peritoneum. Strict aseptic precautions have succeeded in greatly reducing, but not entirely eliminating the danger from this source in all operations requiring opening of the free peritoneal cavity. In genuine cases of diffuse septic peritonitis surgical intervention is usually powerless to prevent speedy death from toxæmia.

(*c*) *Perforative peritonitis.* Perforative peritonitis invariably occurs as a secondary affection usually in connection with an ulcerative or gangrenous lesion of any part of the gastrointestinal canal. Perforative ulcer of the stomach or duodenum, or typhoid or tubercular ulcers of the ileum, perforation or sloughing of the appendix, the different forms of intestinal obstruction, are the most frequent cause of this well-defined clinical form of peritonitis. Perforative peritonitis is manifested by the sudden onset of the disease, by diffuse pain and tenderness, rigid abdominal walls, fever, vomiting, the impossibility by inspection, palpation, and auscultation to ascertain intestinal peristalsis, the latter being almost positive proof of the presence of gas in the free peritoneal cavity. According to the author's observations, meteorismus peritonei in perforative peritonitis caused by affections of the appendix is rare, while he has seldom found it absent in perforations of any other portion of the gastrointestinal canal. According to the number and virulence of the microbes which find their way into the peritoneal cavity with the extravasation, the resulting peritonitis is either diffuse or more or less circumscribed. The colon-bacillus is invariably present in the inflammatory product, but in addition streptococci, staphylococci, putrefactive bacilli, the typhoid bacillus, or bacillus of tuberculosis, according to the nature of the primary infection, may also be found.

Perforative peritonitis must be regarded and treated as a strictly surgical disease. The primary lesion must be exposed and treated as soon as a diagnosis can be made and the necessary measures applied to limit the extension of the infection and to prevent death from toxæmia.

(*d*) *Circumscribed peritonitis.* A circumscribed peritonitis is an inflammation of the peritoneum during which a greater or less part of the peritoneal cavity becomes excluded from the original source of infection by the formation of plastic, visceral, parietal, or visceral and parietal adhesions. The complexus of symptoms varies according to the degree of virulence of the microbic cause, which only occasionally is overshadowed by the primary affection. The symptoms appear suddenly or are preceded by those incident to the primary disease. The severity of the pain and the extent of the muscular rigidity and tenderness will correspond with the extent of the disease. The intensity of the general symptoms is determined more by the nature and virulence of the microbic cause than the size of the peritoneal surface involved. The inflammatory focus may be limited to a very small space, or it may involve the greater portion of the peritoneal cavity and organs which it contains. The clinical course and termination are determined largely by the nature and virulence of the primary bacterial cause, the anatomical location of the primary starting-point, and nature of environment. If the organs adjacent to the primary focus of infection are favorably located to limit the process, diffusion is frequently prevented by the formation of adhesions. This is especially true in cases where the primary infection is limited by the existence of old adhesions.

Localized peritonitis may be confined to the lesser peritoneum, particularly in cases of perforating ulcer of the stomach. More frequently it is caused by appendicitis and cæcitis. A very frequent cause of circumscribed peritonitis is inflammation about the gall-bladder, uterus, Fallopian tubes, and ovaries. The localized form of peritonitis is very often overlooked during life. It can usually only be detected if a demonstrable swelling forms at the seat of inflammation. The mildest form of infection gives rise to fibrinoplastic peritonitis, which leaves temporary or permanent adhesions, but terminates without pus-formation. Circumscribed suppurative inflammation is always attended by fibrinoplastic peritonitis; the products of which and the viscera which it involves form the abscess-wall. The microbes which produce most frequently fibrinoplastic peritonitis without suppuration are the gonococcus and the staphylococci. Circumscribed suppurative peritonitis is usually the result of infection with staphylococci, bacillus coli commune, and pneumococci. In fibrinoplastic peritonitis surgical interference becomes only necessary when intestinal obstruction is caused by the adhesions. In circumscribed suppurative peritonitis the pus should be evacuated as soon as the disease is recognized, and, if possible, by an extraperitoneal route.

(*e*) *Hæmatogenous peritonitis.* In very rare instances peritonitis occurs without any injury or discoverable antecedent lesion of any of the abdominal or pelvic organs, and is then described as idiopathic peritonitis. As peritonitis is always caused by bacteria of some kind, a peri-

tonitis that develops independently of a local source of infection is the result of an infection through the blood, and should be called hæmatogenous or metastatic peritonitis. It has been observed in connection with nephritis, pyæmia, rheumatic arthritis, and acute exanthematous diseases.

(*f*) *Visceral peritonitis.* A localized peritonitis that can be brought in direct etiological connection with the organ primarily affected is expressed by a compound word with the prefix peri and the noun used to indicate the organ primarily affected in a state of inflammation. Thus the anatomical forms of peritonitis present themselves:

Perigastritis,
Perienteritis,
Perityphlitis,
Periappendicitis,
Pericolitis,
Perihepatitis,
Perisplenitis,
Pericystitis (urinary and gall-bladder),
Perimetritis,
Perisalpingitis, and
Perioöphoritis.

As the mesentery and omentum are only duplications of the peritoneum, we have to add to the above anatomical forms mesenteritis and epiploitis.

(*g*) *Pelvic peritonitis.* Pelvic peritonitis is seldom met with in the male. It is a form of peritonitis in which the female pelvic organs are the primary starting-point of infection, with extension to the peritoneum, either through the Fallopian tubes or the lymphatics of the uterus or its adnexa. It is caused most frequently by gonorrhœal or puerperal infection, or develops after instrumental examination of the interior of the uterus or operations upon this organ.

(*h*) *Puerperal peritonitis.* By the term puerperal peritonitis is understood a progressive inflammation of the peritoneum occurring in consequence of an extension of an infection from any part of the genital tract in puerperal women after delivery or abortion. The infection usually takes place through the lymphatics, and in the majority of cases terminates in diffuse septic peritonitis.

(*i*) *Subdiaphragmatic peritonitis.* A peritonitis limited to the under surface of the diaphragm and any of the adjacent organs is called subdiaphragmatic peritonitis. If the inflammation remains limited, and life is sufficiently prolonged, it usually terminates in the formation of a subdiaphragmatic abscess.

SYNOPSIS OF CLASSIFICATION OF ACUTE PERITONITIS.

ANATOMICAL . .	Ectoperitonitis, Endoperitonitis, Parietal peritonitis, Visceral peritonitis, Mesenteritis, Epiploitis, Perigastritis, Perienteritis, Perityphlitis, Periappendicitis, Pericolitis, Perihepatitis, Perisplenitis, Pericystitis (urinary and gall-bladder), Perimetritis, Perisalpingitis, Perioöphoritis, Pelvic peritonitis, Diaphragmatic peritonitis.
ETIOLOGICAL . .	Traumatic peritonitis, Idiopathic peritonitis, Perforative peritonitis, Metastatic peritonitis, Puerperal peritonitis, Peritonitis infantum, Fœtal and intrauterine peritonitis, Peritonitis neonatorum.
PATHOLOGICAL . .	Diffuse septic peritonitis, Putrid peritonitis, Hemorrhagic peritonitis, Suppurative peritonitis, Serous peritonitis, Fibrinoplastic peritonitis.
BACTERIOLOGICAL	Streptococcus infection, Staphylococcus infection, Pneumococcus infection, Bacillus coli commune infection, Gonococcus infection, Tubercular infection.

- Clinical
  - Ectoperitonitis,
  - Diffuse septic peritonitis,
  - Perforative peritonitis,
  - Circumscribed peritonitis,
  - Hæmatogenous peritonitis,
  - Visceral peritonitis
    - Mesenteritis,
    - Epiploitis,
    - Perigastritis,
    - Perienteritis,
    - Perityphlitis,
    - Periappendicitis,
    - Pericolitis,
    - Perihepatitis,
    - Perisplenitis,
    - Pericystitis,
    - Perimetritis,
    - Perisalpingitis,
    - Perioöphoritis.
  - Pelvic peritonitis,
  - Puerperal peritonitis,
  - Subdiaphragmatic peritonitis.

TROPICAL ABSCESS OF THE LIVER,

WITH A CONSIDERATION OF ITS PATHOLOGY AND CLINICAL HISTORY.

By W. T. Howard, Jr., M.D.,
PROFESSOR OF PATHOLOGY;

AND

C. F. Hoover, M.D.,
PROFESSOR OF PHYSICAL DIAGNOSIS IN THE WESTERN RESERVE UNIVERSITY, AND VISITING PHYSICIAN TO THE CLEVELAND CITY HOSPITAL.

The observation, in two small medical services in four months, of four cases of a disease which has been hitherto unrecognized in Cleveland has seemed to us worth calling attention to. It is not improbable that, now that attention is directed to the subject, the disease will be found to be of not uncommon occurrence.

Of our four cases two gave histories of previous chronic diarrhœa, the remaining two had diarrhœa on admission, one acute and the other chronic. In the cases with diarrhœa we did not have an opportunity of examining the stools. Three cases had chronic liver-abscesses, which in two communicated with the right pleural cavity through the diaphragm. In the case with diarrhœa of recent date (Case I.) the abscess was evidently acute. In two cases the clinical diagnosis was established by autopsy, in one case by both aspiration and operation,

and in one (Case I.) by aspiration. The amœba coli was found in two cases: in one in the evacuated pus (Case II.), and in one at the autopsy (Case IV.). In Case I., by a mistake, the pus was lost before a microscopical examination could be made. In Case IV. amœbæ were found in the liver-abscess in the right pleural cavity and in the pericardial cavity. In this case there was a triple bacterial mixed infection. In Case I. there was evidently a severe bacterial mixed infection, probably streptococcus. It is much to be regretted that an autopsy could not be obtained in this case. There can be no doubt that all these cases are to be regarded as cases of amœbic liver-abscesses secondary to dysentery. In this opinion we are supported by the occurrence of previous chronic diarrhœa in two cases, existing diarrhœa in two cases, the finding of the scars of healed ulcers in the colon of two cases coming to autopsy, and the demonstration of the amœba coli in two cases. All of the patients lived in Cleveland.

Case I.—Man, aged thirty-six years, laborer, entered the Lakeside Hospital (service of Dr. Powell) May 26th, giving a history of having been severely ill only five days before admission to the hospital. He complained of chills, fever, and profuse sweating and pain in the right hypochondrium near the border of the ribs. Diarrhœa at present not severe; two soft movements daily, containing a large amount of mucus. Has had former attacks of severe diarrhœa; no blood in the stools. The patient suffered from daily chills and profuse sweating, with rapid rises and remissions of temperature.

On June 2d, at 3 A.M., the temperature was 106° in the axilla; at 8 A.M., 93° in the axilla. The frequent chills and rises in temperature, coming at irregular intervals of from twenty-four to forty-eight hours, persisted up to the time of death, which occurred on June 20th.

An autopsy was forbidden. Exploratory puncture in the right hypochondrium, between the ninth and tenth ribs in the anterior axillary line, yielded a pint of grayish-red pus resembling anchovy sauce. Unfortunately, through a mistake, the pus was lost, so that a microscopic examination of it was not made.

For the clinical history of the following case we are indebted to Dr. W. Rogers, with whom the patient was seen in consultation:

Case II.—O. E., laborer, aged thirty-two years, admitted to the City Hospital May 11, 1895. The patient has been a resident of Cleveland for five years. One and one-half years ago he had a severe diarrhœa with colic and bloody stools. The diarrhœa persisted for two months. During last summer (1894) he had "pneumonia." For the past eight months he has been in a moderately good condition, until a month before admission to the hospital, when he was taken sick with diarrhœa, bloating of the abdomen, and nausea. About the same time a stitching pain in the lower right thorax in the axillary line appeared. At present the diarrhœa is not severe. The patient is very weak, has no appetite, has frequent chilly sensations, sweats profusely at nighttime, expectorates large amounts of mucopurulent sputum, marked dyspnœa. Urine acid; specific gravity 1020; no sugar or albumin.

May 14. Urine acid; specific gravity 1010; no sugar or albumin; amount, 61 oz. in twenty-four hours; urea, 216.5 grains in twenty-four hours. From the physical examination is given the following abstract: patient is emaciated; skin light and clear; face dark; conjunctivæ and mucous membrane of mouth slightly icteroid; tongue red at tip and edges, furred in the centre; pulse, small volume, rapid, low tension; thorax moderately broad and deep; abdomen prominent, especially in the epigastrium; the anterior thorax showed nothing pathological in either the heart or lungs. In the supra- and inter-scapular areas there is no essential difference between the two sides. Over the seventh and eighth ribs in the scapular line on the right side the percussion-note is dull, tympanitic in character. From the eighth rib downward the percussion-note shows an absence of resonance. Over the dull, tympanitic area in the right infrascapular region the inspiration and expiration are bronchovesicular in character. From the eighth rib downward no respiratory sounds are audible.

The liver is uniformly enlarged, reaching three fingers' breadth below the border of the ribs. At the costal border in the anterior axillary line of the right side a friction is palpable and audible, accompanying the respiratory excursion of the liver. The spleen is not palpable, but the percussion-area is enlarged. The liver is sensitive to pressure, but the abdomen is nowhere else sensitive to pressure. During the following week the lower border of the liver ascended nearly two fingers' breadth. The patient was rapidly losing in strength.

On May 20th an exploratory puncture in the ninth intercostal space in the posterior axillary line yielded a thin yellow pus which on microscopic examination showed a relatively small number of well-preserved leucocytes. Large cells answering to the description of amœba coli were seen in considerable number, though no amœbic movement was seen.

On May 23d Dr. Hobson drained the abscess from the ninth interspace in the posterior axillary line—fully two pints of pus were evacuated; drainage established.

A few days later all the signs of an empyema appeared. The sweats and fever continued. The patient's strength gradually diminished. Death occurred June 11, 1895. Unfortunately, a complete record of the temperature-curve during the entire illness was not preserved. The temperature reached its highest point gradually about midnight, accompanied by profuse sweating. The highest temperature was 104.4°, a remission of five degrees occurring within six hours. From May 20–23d (day of operation) the temperature gradually descended to 98°. For eight days after the operation the temperature ranged from 97.5° to 99°. The last six days of life a subnormal temperature was constant.

Autopsy by Dr. Hoover. Anatomical diagnosis: a large abscess the size of a cocoanut in the upper portion of the right of the liver, with adhesion of the adjacent portion of the liver to the diaphragm and to the abdominal wall. A sinus admitting the point of the little finger connected the abscess-cavity with the right thorax. Acute empyema (2 quarts of thin, gray pus), compression of the right lung, chronic tuberculosis of the apex of the left lung. Limited chronic fibrous peritonitis over the descending colon and sigmoid flexure, with cicatrices; remains of old ulcers on the corresponding internal surface of the intestine. Chronic passive congestion of kidneys and spleen.

The liver-abscess was filled with a thin, grayish-yellow pus. The

walls of the abscess were irregular and ragged and covered with a friable grayish necrotic material. Outside of this there was a thin zone of relatively firm fibrous tissue. The pus in the liver-abscess and in the thorax presented the same appearances.

CASE III.—J. J., male, laborer, aged thirty-two years; admitted to the City Hospital August 31 1895. Parents living and in good health. Three brothers and one sister dead; causes unknown.

The patient says he has never been sick before excepting from excessive drinking. Patient has never been south of northern Ohio. Former occupation was that of seaman on the Great Lakes. His present illness began a week before admission with a tired feeling; headache; epistaxis (not severe); occasional vomiting; insomnia (says he was delirious at times); night-sweats; no pain in the thorax; slight cough; expectorates a small amount of yellowish sputum; has been losing flesh rapidly; pain in the lower abdomen severe. The patient has frequent stools (seven a day), consisting largely of freshly clotted blood. Microscopic examination for amœbæ was negative; urine acid, specific gravity 1014; sugar, albumin, and diazo negative. The patient was seen first by the visiting physician on September 3d, ten days after the beginning of the illness (according to the patient's own account). The following observations were made at that time: the examination of the thorax revealed nothing pathological. The liver encroached upon the right thorax two fingers' breadth in the axillary and scapular lines, and although the hepatic dulness extended three fingers' breadth below the costal border in the nipple-line, the edge of the liver was not palpable. Pressure under the costal border in the right hypochondrium causes pain. In the midaxillary line over the hepatic area a friction-sound is heard during the respiratory excursion of the liver. The sound is not audible in the nipple-line nor at the costal border, and is not palpable. The spleen is palpable on deep inspiration at the border of the ribs. On the following day (4th) the patient was in a state of collapse, so that an exploratory puncture of the liver was impossible. Death occurred during the morning of the 5th.

An autopsy was forbidden, but the following exploratory punctures were made. The ninth intercostal space in the midaxillary line (right side) yielded a small amount of reddish serum which contained a small amount of cell-detritus and a few red blood-corpuscles. In the ninth interspace in the scapular line a moderately thick bloody pus (anchovy-sauce); microscopically a moderate amount of fat-globules; very few well preserved cells—*i. e.*, pus-cells—could be found. The only cells in a state of preservation were a few fatty degenerated liver-cells.

A third puncture in the eleventh intercostal space, posterior axillary line, gave the same result as above. Exploratory punctures were made in the seventh interspace in the nipple-line, at the eighth costal cartilage on the right side, and over the splenic area, with negative results. The search for amœbæ in the pus was negative.

The temperature was as follows: August 31st, P.M., 100.5°. September 1st, A.M., 99.2°; P.M., 101.2°. 2d, A.M., 98.5°; P.M., 100.2°. 3d, A.M., 97.6°; P.M., 100.8°. 4th, A.M., 97.2°; P.M., 100°. 5th, A.M., 97.4°.

CASE IV.—O. W. H., male, aged fifty-four years; occupation carpenter; admitted to City Hospital July 18, 1895. The patient lived in Cleveland twenty years preceding his death, never having been

more than a few miles from the city during the entire time. There is no history of tuberculosis in his family. He denies ever having had syphilis. Had typhoid fever twenty years ago. Has had occasional "rheumatism of his joints," though there are no traces of arthritis at present visible. Drank large amounts of whiskey until two years ago, since which time he has drunk very little. During the preceding fall (1894) he had an attack of severe diarrhœa accompanied by colic. This lasted about a month; with the cessation of the diarrhœa he noticed that he voided an unusually large amount of urine each day. In a month after the diarrhœa ceased he had largely regained his former strength, and with the exception of occasional attacks of pain in the hepatic region (which were accompanied by dyspnœa) lasting several days at a time, he was in relatively fair condition until two weeks before admission to the hospital, when severe pain in the right hypochondrium and inferior right thoracic region set in. The pain has been persistent; shortness of breath has also been annoying. While going about he has noticed that the dyspnœa increased toward evening, when he noticed occasional slight swelling about the ankles. Patient has slight cough, with slight mucous expectoration. There are two to four loose movements of the bowels a day.

Urine: specific gravity 1020; acid; no albumin or sugar. Physical examination made on July 25, 1895: development good; nourishment poor; small amount of panniculus adiposus; skin harsh and dry; yellowish pigmentation of entire skin. Both inguinal glands and the left axillary glands are about the size of a small hazelnut. No icterus; slight cyanosis. Dilatation of peripheral vessels of both cheeks; no œdema; no tenderness or thickening of the long bones. The right lateral vein of the thorax is dilated. Immediately above the umbilicus is a slight protrusion the size of a ten-cent piece which feels much like a coil of veins (caput Medusæ). The respiration is largely thoracic in character and increased in frequency. Thorax is moderately broad and deep. The right side in the inferior area is slightly retarded in the respiratory excursion, the difference in the two sides being very slight.

The intercostal spaces in the right hypochondrium are flattened out; in the left side they are retracted. Heart's apex-impulse is visible in the fourth interspace in the nipple line. Pulse has a moderate rate, is rhythmic, regular, small wave; short duration; easily compressible. Atheroma of both radials is pronounced.

Percussion: upper border of the heart is at the third rib in the left parasternal line; left border of the heart in the nipple-line, the right border at the left border of the sternum. The percussion-note in the right supraclavicular space is slightly less resonant than in the left. In the infraclavicular and second interspaces on the two sides there is no essential difference. There are increase of resistance and diminution of resonance toward the base on the right side. The lower border of the lung on the right side is on the fourth rib in the nipple-line, and the fourth interspace in the axillary line; respiratory excursion is present, though diminished in degree. The lower border of the left lung in the axillary line is on the seventh rib.

Posterior thorax: on the right side there are slight increase of resistance and diminution of resonance toward the base of the lung. The lower border is on the eighth rib in the scapular line; on the left side the lower border of the lung is at the tenth rib in the scapular line.

The respiratory sounds are relatively loud over the entire pulmonary area, excepting at the right base, where the intensity of the respiratory and expiratory sounds is much diminished. Nowhere are there any adventitious sounds audible.

Abdomen is protruding, everywhere tympanitic; no free fluid. The right hypochondrium is sensitive to pressure. The hepatic dulness is seven inches in the nipple-line and only two fingers' breadth below the border of the ribs. In the axillary line it measures eight inches. A distinct hepatic border is not palpable, though the movement of the liver on respiration is visible below the costal border. The abdominal wall is tense and resistant.

Splenic dulness is at the eighth rib in the axillary line and extends forward to within two fingers' breadth of the costal border. The spleen is not palpable on deep inspiration.

Hemorrhoids are present; no bleeding at present. The condition of the patient remained much the same without any notable change during the month of August. Being away on a vacation during the latter part of August and first week in September, I was surprised to find on September 6th that the dyspnœa had increased, and that from the clavicle downward on the right side the thorax was flat on percussion, with total absence of vocal fremitus and respiratory sounds. The flat percussion-note reached as far as the left border of the sternum above the precardial area. Three pints of bloody pus were aspirated from the right thorax, after which the upper half of the sternum became resonant. The upper right thorax gave slight resonance on percussion and the respiratory sounds were faintly audible.

From this time the patient failed rapidly. He protested against a second aspiration of the thorax. The fluid again increased in quantity. Death occurred on the morning of September 21st.

The temperature during the first five days gradually fell from evening exacerbations to 101° and morning remissions to 99° to a continuous normal temperature. After the empyema was discovered (ten days before death), the temperature was as follows: September 11th, P.M., 99.2°. 12th, A.M., 98.5°; P.M., 99.8°. 13th, A.M., 96.4°; P.M., 99.2°. 14th, A.M., 96.4°; P.M., 99°. 15th, A.M., 96°; P.M., 95°. 16th, A.M., 95°; P.M., 94.8°. 17th, A.M., 95°; P.M., 95.2°. 18th, A.M., 94.8°; P.M., 94.8°. 19th, A.M., 94.6; P.M., 94.6°. 20th, A.M., 94°; P.M., 95.6°.

The pus aspirated from the thorax was examined for amœba, but none were found. From the history of the patient for the interval during which he was not under my observation it was impossible to determine the date of the origin of the empyema.

Autopsy by Dr. Howard. Anatomical diagnosis: abscess of the right lobe of the liver, with perforation of the diaphragm and empyema of the right side. Chronic pleuritis with retraction of the right lung. Chronic obliterative pleuritis of the left side. Slight chronic morbus Brightii. General heart hypertrophy and dilatation. Acute fibrino-purulent pericarditis. Scars of old ulcers in the colon. Chronic passive congestion of the spleen, kidneys, stomach, and intestines.

Autopsy eight hours after death: body still warm, 170 cm. long, very much emaciated. Subcutaneous fat and muscles wasted. There are no bedsores. The lower portion of both tibiæ rough and irregular, and the skin here of a reddish-brown color. There is no jaundice; the skin is very pale.

The right chest and hypochondrium much larger than the left. The abdomen is not distended.

The peritoneum is everywhere smooth, moist; no excess of fluid. The right lobe of the liver extends 5 cm. below the costal margin in the mammary line. The outer border and a considerable portion of the anterior surface of this lobe are bound by firm adhesions to the adjacent abdominal wall. The left lobe is larger than normal. The upper border of the right kidney is firmly adherent to the lower surface of the right lobe.

Chest: both sides free from œdema. The muscles are markedly wasted and pale. On removing the sternum a large amount of blood-stained, thin fluid escapes from the upper portion of the right pleural cavity. The outer layer of the pericardium is firmly bound by adhesions to the adjacent borders of the lungs. The pericardial cavity contains about 100 c.cm. of blood-stained pus. Both the epi- and pericardium are covered with a layer of fibrous material of varying thickness, which is thickest over the surface of the right ventricle. It is readily removed with the finger, and the underlying membrane is congested and shows many points of ecchymosis. The heart weighs 375 grammes. The wall of the left ventricle averages 17 mm. in thickness. The heart-muscle is pale and tears with ease. The valves and the coronary arteries are normal. The aorta is somewhat dilated.

The left lung, firmly bound down by old adhesions, is voluminous, deeply pigmented, and on section œdematous. It is free from tuberculosis. The bronchial glands are pigmented.

The right pleural sac is distended with fluid. In the upper portion of the cavity there is about half a litre of thin, blood-stained fluid. Below this the rest of the cavity is filled with a thick, brownish-red, anchovy-sauce-like pus containing small, grayish, gelatinous-like masses of tissue.

Both layers of the pleura are everywhere covered with a thick layer of fibrinous material. On the lower portion of this cavity the costal and visceral pleuræ, as well as the diaphragmatic layer, are very much thickened. The lung is flattened out against the posterior portion of the chest, and is firm, dense, and airless, and on section is carnified in appearance. There is no communication between the bronchi and the pleural cavity. The bronchi, veins, and arteries are of ordinary appearance.

Through an opening in the central portion of the diaphragm, large enough to admit the hand, the right pleural cavity communicates with an abscess-cavity in the upper portion of the right lobe of the liver.

The lung, pleura, diaphragm, liver, and right kidney removed *en masse*.

On section the greater portion of the right lobe of the liver is found to be the seat of an abscess, which at its lower posterior portion nearly reaches the surface of the organ. At the lower border of this lobe the abscess again reaches the surface, and at this point the upper border of the right kidney is firmly adherent to the surface of the organ. Just above this the transverse colon is adherent to the adjacent surface of this lobe by rather easily broken-down recent adhesions. There is no apparent relation between the colon and the liver-abscess. The thickness of the hepatic tissue between the wall of the abscess and the surface of the organ varies in different places from 0.5 to 7 cm., the thinnest places being near the upper and the posterior surfaces.

The wall of the abscess is in general ragged and irregular. The superficial layer is soft, easily broken down under the finger, grayish-white in appearance, and varies from 0.5 to 2 cm. in thickness. Beneath is a firmer zone of a peculiar gelatinous appearance and consistency. Outside of this there is a fibrous tissue zone of a varying thickness. The liver-tissue about this is pale and firm. The left lobe is larger than ordinary. The surface is smooth; the capsule is not thickened. On section the surface is pale and has a nutmeg appearance. The consistency is not increased. No other abscess was found. The liver measures 28 x 20 x 16 cm.

The spleen is considerably enlarged and weighs 350 grammes. The surface is smooth and free from adhesions. The capsule is not thickened. On section the tissue is soft and dark red in color. The trabeculæ are visible; the Malpighian bodies are obscured.

The kidneys are of about the same size and general appearance, and somewhat smaller than ordinary. The capsules are lightly adherent in places. On the surface of the left there are several small cysts. The cortices are somewhat thinned. The consistency is not specially increased. The arteries are normal. The renal pelves, the suprarenal bodies, bladder, penis, and testes are normal. The pancreas is normal, as are the pharynx and œsophagus. The mucous membrane of the stomach and small intestine is deeply congested. There are no scars or ulcers in the small intestine. In the rectum, sigmoid flexure, and the ascending colon there are numerous firm scars, but no fresh ulcers. About several of these scars the muscular coat is thinned.

Microscopical examination of the pus from the right pleural cavity, the liver-abscess, and from the pericardium shows large numbers of amœbæ, many of which are actively motile. They vary in size from 30 to 40 and even 50 μ in diameter. In the finely granular protoplasm of many a nucleus can be made out; many contain red blood-corpuscles and pigment; few contain bacteria. Scrapings made from the pleura and pericardium show large numbers of polymorphous nuclear leucocytes, many of which contain fine fat-drops.

In the pus from the liver-abscess no leucocytes are seen. There are a few red blood-cells, some markedly fatty liver-cells, and a few amœbæ. Most of the material is made up of granular detritus. Scrapings of the wall of the abscess contain more amœbæ than the free pus.

Stained cover-slip preparations made from the liver-abscess and from the pus from the pleura and pericardium show small numbers of diplococci, a short, stout bacillus, and a long, thin bacillus. They are more numerous in the pus from the pleural sac than from the liver-abscess or from the pericardium.

In cultures made from the pus from the liver-abscess and from the pleural and pericardial pus there developed colonies of the colon-bacillus and the staphylococcus pyogenes aureus. The cultures made from the spleen and kidneys remained sterile.

Fresh frozen sections of the heart-muscle show a well-marked fatty degeneration.

Microscopical examination of sections made from various portions of the wall of the liver-abscess and stained in hæmotoxylin and eosin shows in all practically the same characters. The inner zone nearest the abscess-cavity is made up of a layer of varying depth composed largely of a granular structureless material that stains rather diffusely with

both hæmatoxylin and eosin. In this material there are irregularly scattered areas composed of small round cells with round or oval nuclei, and a smaller number of spindle-shaped cells. The round cells predominate. The cells extend in some places deep into the underlying tissue, but in other places they are confined to the margin of the abscess-wall. In this zone, in its superficial portion, here and there a few large cells can be made out—amœbæ. Beneath this zone of necrosis there is a broad band of fibrous tissue very poor in cells. In places the necrotic zone extends into this layer. In the outer portion of the fibrous tissue layer there are a few bands or single rows of elongated liver-cells to be seen. There are here and there also many small bile-ducts made out. Outside the fibrous tissue zone the liver-cells are compressed and have lost their shape. The acini can nowhere be made out. The liver-cells are very much elongated and flattened. They contain large amounts of pigment. Just under the fibrous tissue zone many have lost their nuclei and stain poorly. The capillaries are small and often cannot be made out. The portal systems are prominent and the connective tissue about them is markedly increased. Scattered in the liver-tissue there are larger and smaller bands of fibrous tissue. The smaller bands contain a large number of small round cells and the neighboring liver-cells stain poorly, and many of them have lost their nuclei. In sections that reach the surface of the liver the capsule is seen to be markedly thickened and the compression of the liver-tissue extends throughout the sections. There are no areas of definite necrosis or of abscess-formation in this zone of liver-tissue about the abscess-wall. No amœbæ are to be seen. In some sections where the margin of liver-tissue between the abscess-wall and the surface is thin there are small areas of necrosis of liver-cells to be seen. These areas stain diffusely with eosin, and are composed of a hyaline structureless material. In some places there is a diffuse growth of connective tissue, chiefly in the perivascular spaces along the capillaries between the liver-cells. Here the liver-cells are few, and those remaining form flattened elongated bands. The capillaries contain little blood, but the larger portal ones are congested.

Sections stained in methylene-blue and eosin give a clear picture of the abscess-wall. The granular material in the necrotic zone stains diffusely with the eosin. In places along the inner border there are seen numbers of cells, often in clumps, but here and there scattered cells are seen. The most part of these cells are small round cells with single round nuclei and spindle-shaped cells; but here and there masses of polymorphous nuclear leucocytes are seen. In some places numerous red blood-cells are found. In the necrotic material along the free margin of the abscess-wall and sometimes deeper in the tissue there are numbers of bacteria. The most numerous are short, stout bacilli with rounded ends, lying scattered and in masses. Some longer, thinner bacilli are also seen. These also often lie in masses, usually near the surface of the necrotic layer. Scattered in the necrotic material, usually in the areas of leucocytic infiltration, diplococci are seen. These are less numerous than the other forms. The bacteria in some places are found at the border between the necrotic material and the fibrous tissue zone of the abscess-wall.

In one section the epithelium of a large bile-duct is swollen, desquamated, and the lumen is nearly filled with cells.

In the sections stained with methylene-blue and eosin amœbæ can be clearly made out. They are more numerous in some sections than in others. They are only found in the necrotic layer, and are nowhere in great numbers. They vary in size from 10 to 25 μ. They are usually round or oval in outline. Some are stained deeply with eosin, the nucleus being stained somewhat more deeply than the rest of the organism. Others stain rather diffusely but lightly with methylene-blue, the nucleus being stained a darker blue than the rest. In many of the amœbæ vacuoles are seen. At no place can amœbæ be found invading the fibrous tissue zone of the abscess-wall, and they are not found in the capillaries or veins of the liver-tissue.

Pleura: the changes in the pleura are of a much more active inflammatory character than those met with in the liver-abscess. The pleura is covered with a thick exudation, very rich in fibrin and polymorphous nuclear leucocytes. Some of the cells are broken down and have lost their nuclei. In places there are numbers of deeply staining nuclear fragments to be seen. In sections stained by Weigert's method and in methylene-blue and eosin many bacteria are seen. They are mostly diplococci. Besides these there are short, stout bacilli, and longer bacilli similar in size and shape to those described in the wall of the liver-abscess. They are met with chiefly on the surface of the exudation. The diplococci can be traced deep into the exudation and reach the pleura proper. At the border between the exudation and the underlying pleural tissues in most places the pleural cells are absent, and the tissue is infiltrated with small round cells and polymorphous nuclear leucocytes. In places the pleural cells have proliferated, and numbers of them can be seen in the deeper layers of the exudation. The bloodvessels of the subpleural tissues are markedly dilated and congested. In the underlying lung-tissue the air-cells are collapsed and their walls lie in juxtaposition. The lung-tissue is congested and here and there are seen masses of pigment. On and near the exudation a few sparsely scattered amœbæ can be made out. They are not found deep in the exudation or near the pleura. None can be found in the lung-tissue.

Pericardium: sections made from the pericardium over the surface of the right ventricle, and stained with methylene-blue and eosin, show the presence of a fibrinous exudation. In this there are not a great number of leucocytes. The bloodvessels of the endocardium are deeply congested. In the exudation large numbers of cocci are seen. Besides, there are a few long bacilli and some short, stout bacilli. In places the lining cells of the epicardium are swollen and desquamated. In the exudation, besides these cells, there are larger ones, staining like the amœbæ in the liver-abscess and of the same size and appearance. They can be distinguished from the epicardial cells by their size, the presence of larger and smaller vacuoles, and the difference in their nuclei. They are fairly numerous. The epicardial cells are smaller, have no vacuoles, and have larger, rather oval vesicular nuclei.

In the hardened specimens no special changes are seen in the heart-muscles.

The kidneys show well-marked chronic passive congestion and slight chronic diffuse nephritis. The spleen is markedly congested.

PATHOLOGY. In considering the pathological anatomy of tropical abscess of the liver, we will deal first with that form associated with

previous or existing dysentery, and then with the so-called "idiopathic" liver-abscess. We shall leave out of consideration entirely the metastatic liver-abscesses following typhoid and tuberculous lesions of the intestinal tract, catarrhal and croupous dysentery, peritonitis, appendicitis, and perityphlitis, and pyelitis of the portal vein, suppurative endocarditis and the like. All these varieties are usually seen as multiple small abscesses, and are quite distinct in their etiology, size, situation, and anatomical appearances from the tropical abscess.

The form of dysentery followed by liver-abscess is peculiar, etiologically and anatomically. It is characterized by the formation of larger and smaller ulcers, usually deep, with irregular overhanging edges. In many places, in addition to the ulcerative process, larger and smaller abscesses exist in the submucosa under an apparently intact mucosa. In some places these communicate with the lumen of the intestine by small openings, through which puriform material can be squeezed. In some cases the muscularis is extensively invaded, and perforation may result. In the more severe cases there are long sloughs involving the mucosa, submucosa, and muscularis. Kartulis, Councilman and Lafleur, and Kruse and Pasquale, especially, have identified this alterative form of dysentery with the tropical dysentery and have established the etiological relation of the amœba coli to it. These authors and others have found this organism invariably associated with the lesions in a way that leaves no doubt that it is the cause of the ulcerative process, which may be and usually is modified by the presence of various bacteria which mark the ulcers secondarily. The amœbæ penetrate the mucosa and seek the submucosa. They may sometimes be found in the intestinal glands. The primary changes are in the submucosa. The mucosa is affected secondarily. Abscesses of varying size are found in the submucosa, and may burrow for considerable distances without breaking through the mucosa or invading the muscularis. Usually, however, both of these coats are finally involved in the process. Amœbæ are present in large numbers in these abscesses and in the ulcers. The changes are usually limited to the colon, but not uncommonly the rectum, cæcum, and even the ileum for a short distance are involved. The tissue-changes are chiefly those of necrosis and liquefaction. The amœbæ are abundant in the lesions, and may be found for some distance away penetrating into the neighboring tissues.

The proportion of cases of this amœbic dysentery complicated with liver-abscess varies within rather wide limits in the experience of individual observers. The statistics collected by Councilman and Lafleur show that in India in 1429 autopsies on individuals dying of dysentery, liver-abscess occurred in 306, or one case of liver-abscess to every four and one-half cases of dysentery. In Algiers, of 1001 autopsies on dysentery cases 180 had liver-abscess, or a proportion of one liver-

abscess to five of dysentery. According to Kartulis, of 500 cases of dysentery, from 50 to 60 per cent. had liver-abscess. Of 40 American cases collected by us, 18 had liver-abscess. Liver-abscess may occur in the acute form of dysentery, but it is more common in the chronic variety, and then its course is often so insidious that clinical symptoms are lacking until the abscess is large or even ruptures.

By far the most common seat for these abscesses is in the right lobe of the liver. When the left is affected, it is usually in conjunction with the right. They are usually situated in the upper portion of the right lobe of the organ, just beneath the diaphragm. Next in frequency comes the lower portion of the right lobe, corresponding to the hepatic flexure of the colon. Kruse and Pasquale describe in one case an abscess in the lobus quadratus. The abscesses may start near the surface or deep in the hepatic tissue. They are more commonly single, but may be multiple; rarely, however, are there more than two or three. Occasionally they are small and numerous, widely distributed throughout the organ, and affecting both the right and the left lobes. In some cases several small abscesses may be grouped about a large one. Sometimes two or more abscesses may be connected by sinuses.

In size the abscesses vary from microscopic points to large collections of pus occupying as much as two-thirds or more of the liver-substance.

The abscess-contents consist usually of a reddish-brown, anchovy-sauce-like material, which is in some cases of an almost gelatinous consistency. In other cases the material is of a grayish-white appearance and contains larger and smaller masses of necrotic tissue.

The abscess-walls are usually ragged and show larger and smaller, irregular, soft, grayish masses of necrotic material. When the process is of recent date the abscess-wall is soft and the line of demarcation between it and the liver-tissue is irregular. In the older abscesses, beneath the soft necrotic material, there is usually a layer of rather translucent gelatinous-like material of varying thickness. Beneath this, and separating it from the surrounding liver-tissue, there is a zone of fibrous tissue which in old abscesses is dense and firm. This zone varies very much in thickness. In these abscesses the necrotic zone is often narrow and the fibrous tissue layer is thick. Always beneath the fibrous tissue zone the surrounding liver-tissue is compressed and indurated. Not infrequently scars of healed abscesses are found in the livers of individuals who have had dysentery, and die long afterward of some other affection.

Livers the seat of abscesses vary very much in size. Often the organ is not at all enlarged, and it may even be smaller than normal in cases of chronic abscess in the process of healing. Often the abscess or abscesses just fill the space of the degenerated and necrotic liver-tissue. In other cases the liver is enormously enlarged, usually in the case of

multiple abscesses. In chronic abscesses the size of the liver or of the affected lobe is often much decreased, due to the absorption of the purulent material, and the focal and diffuse cirrhosis that follows. The more fluid portion of the abscess-contents is composed almost entirely of granular detritus, and few well-preserved cellular elements are found. In recent abscesses degenerated granular and fatty liver-cells may be seen; in the pus from older abscesses these are usually absent. Rarely are polymorphous nuclear leucocytes found, and when present they are very few in number, and usually there is a bacterial mixed infection. Red blood-corpuscles are commonly seen, both well preserved and in various stages of degeneration. Careful search, which, however, in some cases must be repeated, practically always discloses amœbæ. Often they are missed in aspirated pus, which probably usually comes from the central and older part of the abscess.

In even the smallest abscesses Councilman and Lafleur found amœbæ. According to their studies, the tissue-cells undergo necrosis and finally become liquefied, and this is probably associated with a serous exudation from the bloodvessels. There is a striking absence of nuclear fragmentation. In some cases they describe a necrosis of liver-cells in which the cells preserve their form, while in others they are broken up into highly refractive masses, and in still others they appear as fragments lying between the capillaries. These changes were noted in all of their cases, were not associated with the presence of amœbæ, and were best marked in an acute case. These changes were met with always in the central portion of the lobule, and were often confined to a few cells near the central area. They showed no leucocytic invasion, and were not associated with the complete liquefaction of tissue and the formation of abscesses that occurred when amœbæ were present. These authors attribute these diffusely scattered areas of necrosis of liver-cells to the action of soluble toxic substances produced by the amœbæ in the intestinal lesions, and absorbed and then brought to the liver, while the necrosis and liquefaction of tissue in the abscess-areas are due to the direct action of the amœbæ themselves.

They point out that these focal areas of necrosis may lead to cirrhosis of the parts of the liver affected, as sometimes occurs in India as a sequence of dysentery.

The small abscesses may be microscopical in size. They vary in shape, and their contents consist of firmly granular material with here and there cellular and nuclear fragments, and in some there is fibrin. Few, if any, leucocytes are seen, but red blood-corpuscles may be numerous. In the larger abscesses there may be more solid masses composed of liver-tissue that has escaped destruction. Often these masses represent portal systems, the interportal tissue having liquefied. Amœbæ are always numerous in the smaller abscesses, being more numerous about

the periphery, extending in places into the liver-tissue, but not usually beyond the area of tissue-necrosis. A few may be found in the capillaries. The smaller abscesses do not exert any pressure upon the surrounding tissue. Abscesses of microscopic size may be found in every way connected with the larger abscesses.

In the larger and older abscesses generally three zones can be made out: an inner necrotic granular zone, then a highly refractive reticulum, and outside of this a layer of granulation-tissue of varying thickness. The appearances described in Case IV. are quite characteristic. The inner necrotic zone shows granular detritus, a few or more small round cells, few or many red blood-cells, and sometimes polymorphous nuclear leucocytes. Sometimes necrotic and fatty liver-cells are present. Usually in this zone amœbæ can be made out. They vary very much in number. Some sections may contain many, while in others few or none are seen. A striking thing in most cases is the absence of polymorphous nuclear leucocytes.

The granulation-tissue of the outer zone may be markedly cellular, but in the older abscesses the cells are few in number and those present are, for the most part, spindle-shaped.

Bile-ducts lined with cuboidal epithelial cells are numerous about and often in this layer. The bloodvessels are dilated, but often the walls of the capillaries are thickened. At the margin of the fibrous tissue zone the liver-tissue is compressed, and the liver-cells, being spindle-shaped, often lie in long, flattened rows. The connective tissue of the perivascular spaces is increased, many of the capillaries are obliterated, and the central areas have disappeared. The bile-ducts are numerous in some places and absent in others. There is a more or less well-marked cirrhosis of the liver-tissue, then, for a varying distance from the abscess.

There is a striking difference between the appearances, both in the acute and in the chronic amœbic abscesses, from those met with in abscesses due to bacterial invasions. In the amœbic abscess there is a striking absence of leucocytic reaction, and both the amœbæ and their toxic products evidently lack positive chemotactic properties for leucocytes, or even exert negative chemotaxis on these cells. In the amœbic abscess with bacterial mixed infections the difference in the number of leucocytes present is marked. In general, it can be said that pus with few or no leucocytes coming from a liver-abscess is of amœbic origin. If, however, there is a bacterial mixed infection, numbers of leucocytes may be present, the organisms causing the mixed infection being the cause of the leucocytic invasion.

Liver-abscesses may communicate with various organs and cavities. The most common secondary invasion is undoubtedly through the diaphragm into either the right pleural sac or directly into the lower lobe of the right lung.

If the communication here is due to a sudden rupture of a liver-abscess through the diaphragm, or, as may also occur, a rapid extension of the amœbic microbe process through this organ, the pleura rather than the lung is affected.

The opening of a liver-abscess into the lung, and the extension of the process into this organ, more commonly take place when the process gradually extends through the diaphragm and there is time for the occurrence of an adhesive inflammation between the visceral and diaphragmatic pleura. In the former case there is a sudden rupture through the diaphragm, with the evacuation of a large amount of pus into the pleural sac, with retraction of the lung. The pleura soon becomes thickened and covered with a ragged, easily broken-down necrotic material similar to that lining the liver-abscess. The retracted lung becomes firmly bound down by the thickened pleura, the thickness of which varies greatly. The pleural reaction in these cases usually protects the lung from invasion, and a pneumothorax rarely occurs. When the process from the liver-abscess invades the lower lobe of the right lung the resulting abscess shows very much the same appearances met with in the liver. The histological changes are, according to Councilman and Lafleur, much the same. The changes in the interstitial tissue are, however, more marked in the lung than in the liver, and there is usually a well-marked chronic interstitial pneumonia, the reaction being on the part of the interstitial tissue of the alveolar walls. The same degeneration and liquefaction of tissue are met with in the lung as in the liver. Abscess-formation is usually confined to the lower lobe, and the abscesses are usually single, but may be multiple. The lung-abscesses usually open into one or more bronchi, and the hepato-pulmonary abscess is thus drained. One of us has twice been able to make a diagnosis of unsuspected hepato-pulmonary abscess from an examination of the sputum; in both cases amœbæ were present in large numbers and were by far the most numerous cells in the sputum.

The lung is rarely, if ever, infected with amœbæ by the circulation. If amœbæ are carried from the liver by the hepatic vein—and it is difficult to see why this must not uncommonly happen—they certainly do no harm. Rarely, liver-abscess may rupture into the vena cava, and a large amount of pus may be carried to the lung, but death occurs before multiple abscess can form.

The pericardium may be invaded in two ways: (*a*) by the opening of a liver-abscess directly into the pericardium, and (*b*) by extension from either the pleural cavity or from a lung-abscess, either by rupture or by the gradual extension of the process through the pleura and pericardium, of which Case IV. is an example. A sinus may connect the two cavities. The character of the pus found in the pericardial cavity will depend upon the source of the pus: whether it is from the liver, the

pleura, or the lung; upon the length of time the pericardium has been affected; and upon the presence or absence of a bacterial mixed infection. In our case with mixed infection there was a marked inflammatory reaction, with the presence of a number of leucocytes in the pus. Amœbæ have not, so far as we have been able to learn, been described in the pericardium. In Case IV. they were found in the fresh pus, in the necrotic material covering the pericardium, and in the sections of the hardened tissues.

Hepatic abscesses may rupture into the gall-bladder and discharge their contents into the duodenum, or they may rarely rupture into the stomach.

Rupture into the abdominal cavity occurs but rarely. The right kidney may form a part of the wall of a liver-abscess and the pus may discharge in the urine.

Thierfelder collected 170 cases of liver-abscess, of which 76 opened into the lung and bronchi, 23 into the abdominal cavity, 32 into the intestine, and 13 into the stomach.

Aghetti collected 131 cases, of which 38 broke into the lung.

Three of our cases opened into the right pleural cavity. By far the most common place for rupture of hepatic abscess is into the thoracic cavity.

In four cases in which the heart-muscle has been examined (three cases by Flexner and our own case, IV.) fatty degeneration has been found. The changes in the kidney are limited to cloudy swelling and fatty degeneration, except in the rare cases where the right kidney forms a part of the wall of a liver-abscess. The spleen is usually enlarged, and is sometimes soft and sometimes firm, the latter usually being dependent upon a long-standing passive congestion. In one case of liver-abscess Krause and Pasquale found small pus-collections in the spleen distributed along the branches of the splenic veins. They think that this was brought about by the backward flow of pus which gained access to the portal vein.

Councilman and Lafleur failed to find the focal areas of necrosis in other organs than the liver. They were not present in our case.

The peritoneum may be invaded in several ways. In a few cases there is a peritonitis starting from the peritoneal coat of the intestine corresponding to the seat of the ulcers. The exudation is sometimes abundant, is of a peculiar gelatinous consistency, and contains large numbers of amœbæ. Bacteria may or may not penetrate the intestinal walls in these cases. If a liver-abscess ruptures into the peritoneal cavity, there is a widespread peritonitis with pus of the same character described above. Chronic fibrous peritonitis with thickening over the ulcers in the intestine is not uncommon. Chronic adhesive peritonitis about the liver, between the organ and the diaphragm, abdominal wall, omentum, and the transverse colon is common.

The macroscopical appearances of the so-called "idiopathic" liver-abscess are described as being similar to those of the chronic abscess associated with dysentery. They usually have firm fibrous walls.

So far as we can learn, there is no account of the histological lesions and no good description of the process. The histological study of the process in these cases promises much in clearing up their etiology.

(To be continued.)

HABITUAL OR RECURRENT DISLOCATION OF THE SHOULDER.

By Herbert L. Burrell, M.D.,
and
Robert W. Lovett, M.D.,
of Boston.

Habitual or recurrent dislocation of the shoulder, although on the whole a surgical rarity, occurs often enough to have attracted to itself a certain amount of theoretical and practical consideration. The name "habitual dislocation" is intended to refer only to those cases where dislocation occurs from causes too slight to displace the normal joint. A person may dislocate one shoulder two or three times from a succession of severe accidents without falling into this special pathological class.

Pathology and Etiology. The pathological condition present varies widely; certain abnormalities have, however, been demonstrated, chiefly in the head of the humerus, where it has been removed by excision for relief of the disability.

Cramer (*Berl. klin. Woch.*, 1882, p. 21) excised the head of the bone in an epileptic woman, thirty years old. The head of the humerus showed a depression in the posterior part of its articular surface. This was 4 cm. long, 2 cm. broad, and $\frac{3}{4}$ cm. deep. There was also found in the joint a loose body with a smooth surface, composed of bone, fibrous tissue, and cartilage, and this loose body was attached by a long pedicle to the back of the glenoid fossa. It seemed to be a piece broken out of the head of the humerus. Dislocation was apparently favored by this abnormality in the shape of the head of the bone.

Volkmann (*Cent. für Chir.*, 1883, p. 28), quoted by Popke, resected the shoulder-joint in a man thirty years of age, an epileptic, whose shoulder slipped out with such ease that the arm was almost useless. The posterior third of the head showed a smooth surface not covered by cartilage, and the glenoid cavity was narrower below than above. The capsule was torn away from the glenoid cavity, and the joint, therefore, communicated with the bursa under the subscapularis muscle. A loose body was also found in this joint, evidently of bony origin, and apparently due to a fracture of the glenoid cavity.

Küster (*Beil. zum Cent. f. Chir.*, 1882, p. 73) excised the shoulder in a young man whose original dislocation was produced by the fall of a

heavy box on the shoulder. The head of the humerus showed a similar loss of substance, but no loose body was found in the joint.

In the discussion following the report of these cases Kraske reported a case operated on at Halle, where the changes in the head of the bone were similar to those mentioned above, and a loss of substance existed also in the glenoid cavity.

The cases of Cramer and Küster, at least, point plainly to a fracture of a part of the joint as a cause of the recurrent dislocations, and that such injuries may occur in simple dislocations of the shoulder is attested by at least three cases (quoted by Cramer):

1. Cooper: subspinous dislocation, subscapularis muscle torn off and attached to the glenoid fossa.

2. Hilton: subglenoid dislocation—greater tuberosity torn off.

3. Deuterlich: both tuberosities torn off and implanted lower down on the humerus.

Löbker (*Beil. zum Cent. f. Chir.*, 1886, p. 90) reported a case, the specimen being obtained post mortem, which seems to belong to another category, and with this goes a similar case resected by Vogt. In each case the anterior part of the head was normal. The posterior half, however, was flattened and showed a depression 1 cm. deep extending from top to bottom. This surface was covered by cartilage, and neither here nor in the glenoid cavity could any evidence of a previous fracture be found. The long tendon of the biceps had been torn from its insertion and was adherent in its groove, and the muscles had apparently been torn from the greater tuberosity.

It does not seem possible to attribute these malformations to fracture, as in the other cases.

Injuries to the muscles and tendons have been demonstrated by Joessel (*Deutsch. Zeitschr. f. Chir.*, 1880, xii. p. 167). He had the opportunity of examining post mortem the bodies of three patients (one with a double habitual dislocation), which furnished four specimens. Analysis of these showed in all cases that the supra- and infra-spinatus muscles had torn loose from the greater tuberosity and had retracted and wasted. By the retraction of the tendons of these muscles the capsule was drawn upon and pulled open so that it communicated with the subacromial bursa. The joint-cavity, therefore, was bounded above by the under surface of the acromion. In one the long tendon of the biceps was ruptured and was adherent to the groove. The cavity of the joint was much enlarged, having in one case a capacity of 90 c.cm., where it should have been only 28 c.cm.

In the two cases operated on by Ricard (*Bull. de l'Acad. de Méd.*, 1894, p. 330) the capsule was found to be thin, flaccid, and lax; no rent was found, nor was the joint-sac opened.

In the case operated on by Gerster (*Rules of Aseptic and Antiseptic Surgery*, p. 8, note 2), and in the two cases operated on by Burrell, no abnormality beyond laxity of the capsule was detected; but it must be

remembered that in these five cases the joint could not be so thoroughly explored and inspected as in the examination of a post-mortem specimen of a resected joint.

A consideration of this pathological evidence (Stimson : *Dislocations*, p. 265, quoting also Gurlt : *Path. Anat. der Gelenk-Kranhheiten*, p. 250 ; Cushing : *Med.-Chir. Trans.*, 1837, p. 336) establishes the probability that in some cases, at least, the cause of the frequent dislocations may be

1. Laxity of the capsule of the joint.
2. Partial fracture of the head of the humerus.
3. Partial fracture of the glenoid cavity.
4. Tearing away of muscular insertions and rupture of tendons.
5. Abnormality in the shape of the head of the humerus not demonstrably due to fracture, but probably the result of chronic, non-suppurative inflammation.

It would seem as if in certain instances the cause of the recurrence of the dislocation was insufficient immobilization of the arm after a primary dislocation. The arm should be immobilized for at least ten days in order that the rent in the capsule of the joint may close.

Symptoms and Diagnosis. Six cases are here presented for analysis, and of these, four have been carefully observed with regard to certain symptoms not heretofore recorded. The other two cases occurred earlier, and were not recorded in these especial symptoms.

In the four cases closely observed, three of the right arm and one of the left, the right shoulder has in all cases drooped.

Muscular Atrophy. Certain of the muscles of the affected side have been so notably atrophied and flabby that they have at once attracted attention on examination. The other muscles have not been notably smaller than those of the other side. The group of muscles affected in these cases has been uniform and constant.

The atrophied muscles are

Coraco-brachialis.
Triceps.
Deltoid, especially the posterior part.
Supra- and infra-spinatus.
Rhomboids.
Levator anguli scapulæ.
Latissimus dorsi.

In a case observed by Lovett, where three dislocations of the right shoulder had occurred inside of a year, each from a sufficiently heavy fall, there was no marked atrophy of these muscles one week after the third accident.

Electrical examination in Cases III. and VI. showed no reaction of degeneration and no qualitative change except in the deltoid, which was more irritable on the affected side (Case VI.).

Limitation of motion was present in all cases to a slight extent, and this limitation was not due to pain nor to fear of displacement, but apparently to some lesion in the joint-mechanism. This was noticed chiefly when the arm was abducted by the patient to a right-angle with the body, and upward swinging of the forearm was attempted. Such rotation in this position was not so free as on the other side. In three of the cases abduction of the arm was limited.

Examination by the fluorescope showed that the kathode rays penetrated the affected shoulder with more difficulty than the other, and at no time was the picture so clear. It could be seen that the head of the humerus was further from the acromion in the affected shoulder. It could be replaced and made to look like the other by raising the point of the elbow. (Cases IV. and VI.)

It should be noted that a large proportion of epileptics are found in all reported cases. Another point of interest is that, although reduction is, as a rule, easy, and inflammatory reaction in the joint is notably slight or even wholly absent after reduction, these patients have a constant and exaggerated dread of dislocation. The condition of "panic" when recurrent dislocation does occur seems to be an analogous mental condition to that of patients with hæmoptysis.

PROGNOSIS. In a shoulder-joint where a dislocation has once or twice occurred from insufficient cause it is not likely that the liability will become less frequent as time advances, if no treatment is undertaken. As a rule, the dislocations will occur with greater frequency and from slighter causes as time progresses.

TREATMENT. The methods of treatment may be classified under four heads:

1. By apparatus.
2. By massage and exercises alone.
3. By temporary fixation and massage.
4. By operation.

1. *Apparatus.* The use of apparatus confining the arm to the side is to be condemned. It weakens the muscles by causing their disuse. It is uncomfortable and partially disabling, and its use can only be considered justifiable temporarily or under exceptional conditions.

2. *Massage and exercises without apparatus.* This treatment is based on the advisability of improving the circulation and nutrition of the shoulder and strengthening the atrophied muscles. From the observations made as to the atrophy of the muscles surrounding the joint, and the relaxation of the capsule of the joint, it is clear that any improvement in the strength of these muscles is logically called for. The shoulder is allowed perfect freedom during this treatment. Case II., treated in this way three times a week for some months, improved in outward appearance, and the muscles, although not regaining their full tone as con-

trasted with those of the other side, grew much firmer. Dislocations, however, occurred as frequently as before this treatment was undertaken.

Case III., on the other hand, treated for six months by the same method, has had no dislocation, having gone longer than his usual period, and the muscles of the shoulder have improved in appearance.

Exercises should be made against resistance, and should be directed to the development by special training of the atrophied muscles. Examples of such exercises would be abduction of the affected arm to contract the deltoid, backward movements from the shoulder, forcible extension of the arm for the triceps, and similar movements.

3. *Fixation in connection with massage and exercises.* Prolonged fixation is called for when a second dislocation has occurred from slight cause. The arm is lifted by applying a sling, which supports the forearm and point of the elbow. The arm is held to the side by a swathe, thus preventing all motions of the joint. This removes as much weight as possible from the joint-capsule.

In connection with this treatment daily massage to the shoulder-muscles should be employed, and especially to the muscles found atrophied in such cases. After some weeks of fixation motion should be progressively allowed along with massage and exercises for the muscles ordinarily affected in these cases. This treatment should continue at least three months. Electricity is of help also in causing special contractions of the atrophied muscles. The faradic current of medium strength should be used.

4. *Operation.* The operative measures advocated have been few. The earliest method advised was to cause an extensive scar in the axilla, thereby binding the arm to the side.

Malgaigne (*Traité des Fractures et Luxations*, vol. ii.) advocated subcutaneous incision of the capsule of the joint by a tenotome introduced anteriorly, in the hope of producing inflammatory contraction.

Removal of the head of the humerus has been done several times, with surprisingly good results as to the restoration of motion and in the matter of usefulness of the arm. Excisions for the relief of this condition are recorded[1] by Cramer, Löbker, Küster, Volkmann, Kraske, and Vogt.

Excision of the head of the humerus is a mutilating operation, and will rarely be necessary. It is conceivable that after an exploration of the joint abnormalities might be found which would demand an excision.

Cramer is recorded as referring to an open incision of the joint as a possible operation in this condition in 1882 (*Berl. klin. Woch.*, p. 21).

Gerster (*Rules of Aseptic and Antiseptic Surgery*, p. 8, note 2) in 1883

[1] Cent. f. Chir., 1883, p. 28: Beilag. z. Cent. f. Chir., 1882, p. 73, and 1886, p. 90; Deutsch. Zeits. für Chir., 1880, xiii. p. 167; Pitha and Billroth, ii. p. 652.

operated upon a recurrent dislocation where the weight of the arm alone was sufficient to displace the joint:

"The joint was freely opened by an anterior longitudinal incision, when it became evident that the tendency to dislocation was due to laxity or redundancy of the anterior part of the capsular ligament. By two semi-elliptical incisions a piece of the capsule one inch long and half an inch in width was removed. The capsular wall as well as the muscular and the skin-wound was united by three tiers of interrupted catgut sutures." Suppuration occurred from improperly kept catgut; the wound was reopened and packed with gauze. No further complication interrupted the course of healing. The habitual luxation was also cured.

In 1892 Ricard (*Bull. de l'Acad. de Méd.*, 1894, p. 334) exposed the capsule of the shoulder-joint by dissection, in the case of a young man, twenty-seven years old, the subject of frequent dislocations of the left shoulder. He did not open the joint-sac, but reefed it anteriorly by stitches passed through its whole thickness about 2 cm. apart. Absolutely normal mobility was secured, and eighteen months later no recurrence of the dislocation had occurred.

A second case was operated on by Ricard a few months later in 1892. The patient was an epileptic, and had had some twenty-eight dislocations. The same operation was performed; restoration of joint-motion resulted, and a year after operation the shoulder had not again been dislocated.

On February 19, 1895, Burrell, through an anterior incision, opened the joint, explored it, removed an elliptical piece of the capsule, and sutured the capsule, thereby shortening it. The history of this case is appended in Case V.

He operated upon a second case February 16, 1897. The history is appended in Case VI.

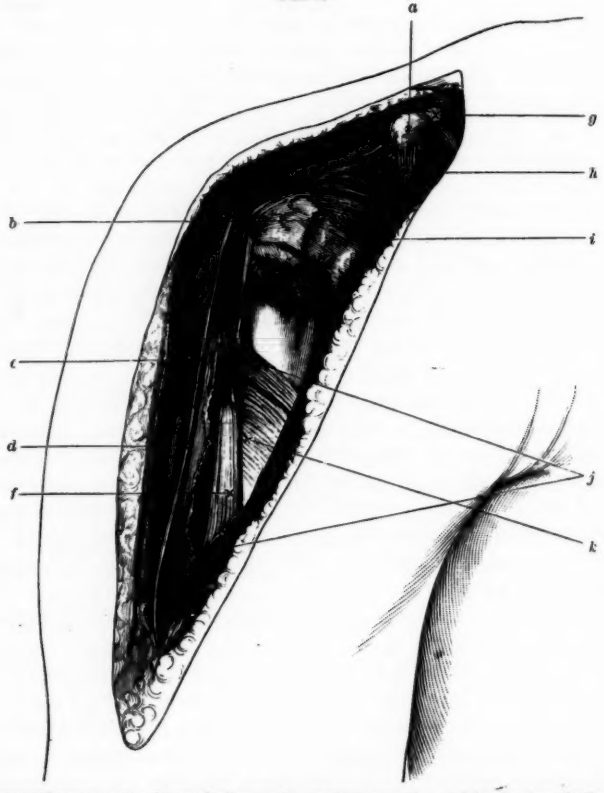
Fig. 1, which shows the field of operation, is a drawing from a dissection kindly made to demonstrate this operation by Dr. F. B. Lund. The important detail which is shown in the cut is the divided tendon of insertion of the pectoralis major. This allows the retraction inward of this muscle, uncovering the capsule of the joint. In order to gain access to the joint the subscapularis muscle should be partially divided. Then with hooked retractors a piece of the capsule can be excised.

The following description gives in detail the operation:

The patient was etherized and the arm slightly abducted. An incision was made from the coracoid process downward and outward, following the line of the cephalic vein to below the upper border of the tendon of insertion of the pectoralis major. The cephalic vein was then recognized, drawn outward, and the intermuscular septum between the deltoid and the pectoralis major was separated with the handle of the scalpel and by a few touches of the blade.

This exposed in the upper part of the wound the coraco-brachialis and short head of the biceps, and in the lower angle of the wound the upper part of the insertion of the pectoralis major.

FIG. 1.



a, coracoid process; *b*, subscapularis muscle covering capsule; *c*, deltoid; *d*, cephalic vein; *f*, long head of biceps; *g*, pectoralis minor; *h*, tendon of coraco-brachialis; *i*, pectoralis major; *j*, edge of divided tendon of insertion of pectoralis major; *k*, tendon of latissimus dorsi.

The acromio-thoracic artery was identified; the upper three-quarters of the insertion of the pectoralis major was divided in order to allow the muscle to be retracted inward and thoroughly to expose the head and neck of the bone. These now came into view, and in front of the head and neck of the bone could be seen and felt, through its sheath, the long head of the biceps.

It was found necessary to clear the tendon of the coraco-brachialis

and short head of the biceps quite up to the coracoid process, and to carry the incision in its whole depth up to the coracoid process.

By externally rotating the arm and dropping it backward the insertion of the subscapularis muscle could be distinguished; its tendon was stretched over the head of the bone. A portion of this insertion was divided. The finger felt the head of the bone, the anterior two-thirds of which was very plainly exposed, and it tended to slip forward toward the coracoid process. The coracoid process could be plainly distinguished, and the capsular ligament was apparently lengthened.

The arm was then abducted to an angle of 45°, and the head of the bone pressed backward to prevent the head from coming up under the coracoid process. By these means the front of the capsule was relaxed. The loose part of the front of the capsule was grasped with three-pronged vulsellum forceps. Three sutures were inserted with a curved needle beneath this, and this fold of the capsule, three-quarters of an inch in length and three-eighths of an inch in width, was excised.

Two of the sutures were cut out at the time of removing the bit of capsule. The other suture was held and tied. Another suture was introduced into the capsule. After these sutures were tightened and tied it was found that the capsule was distinctly tighter and shorter.

The acromio-thoracic artery was divided, and was the only vessel requiring ligature. Sterile water was used for irrigation. Silkworm-gut sutures closed the whole length of the wound. No attempt was made to unite the partially divided insertions of the pectoralis major and of the subscapularis, as when the arm was brought to the side these structures came together without suturing. The wound was dried, an aseptic dressing applied, and the arm fixed to the side with the hand across the chest.

The important steps in the operation are: the free division of the tendinous insertion of the pectoralis major for three-fourths of its breadth, in order that the head of the bone and capsular ligament may be freely exposed by retracting the muscle; the division of a portion of the insertion of the subscapularis; raising the arm to a horizontal plane and pressing back the head of the bone, which relaxes the front of the capsule so that it can be grasped and a bit removed.

A clear differentiation should be made of the anatomical structures, and, with this accomplished, the operation is feasible. The use of a broad retractor, without sharp points, on the inner side of the wound to retract the coraco-brachialis and the vessels is of great importance.

The result of the operation in the first case (Case V.) is that a year and ten months after operation the patient, when seen, stated that there had been no recurrence of the dislocation; that he had been fishing on the Grand Banks during the summer of 1896, and that the motions of the joint were perfect. In the second case (Case VI.), at the end of two

months, the patient is using the arm freely and has had no recurrence of the dislocation.

The first operation took one and three-quarters hours. The second operation took thirty-five minutes. It is purely an anatomical operation, and each anatomical structure must be recognized as carefully as in the ligation of an artery. So far as one can generalize from these two operations it is simple, efficient, and curative.

CONCLUSIONS. The treatment of recurrent dislocations of the shoulder-joint should be by massage and exercise, combined with primary fixation. If this fails after ten weeks' trial, an operation is advisable. This operation should be the exposure of the anterior part of the capsule of the joint, its partial resection, and shortening by sutures.

HISTORIES OF CASES.

CASE I.—A man, aged thirty years, subject to epilepsy, was seen by Dr. Lovett in 1889. The dislocation was subcoracoid, and could not be reduced without ether. It recurred frequently, and was generally caused by epileptic paroxysms at night. The patient only remained under operation for a short time, during which time a bandage was applied, holding the arm to the side. The dislocation was apt to occur whenever the arm was raised from the side. The condition gradually grew worse, and the patient disappeared from observation, being very remiss about treatment.

CASE II.—The patient, a young, healthy, but nervous man, aged thirty-two years, of fair muscular development, was seen by Dr. Lovett October 3, 1896. He had originally dislocated his arm by a heavy fall, and had been treated by an eminent surgeon.

The arm had been kept at rest about ten days, and then he had been allowed the use of it. When seen he had dislocated the arm four times, the last two being upon slight provocation.

He was seen with the arm dislocated in the subcoracoid position; it could not be reduced without ether. The arm was fixed for two weeks, at the end of which time the patient became restive and was unwilling to continue fixation longer. He was warned of the consequences, and was allowed the use of the hand. A few months later he dislocated it in falling down-stairs, and this time he refused to have it done up more than a few days. He experienced little discomfort from the dislocation. He was given massage and electricity, but after a few weeks felt better, and discontinued all treatment. During the summer he went abroad, and dislocated it by a fall of moderate severity.

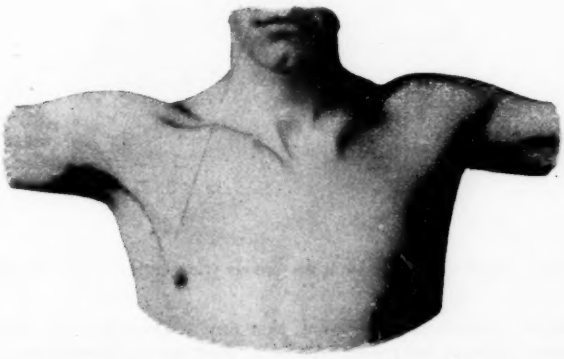
In the fall of 1896 dislocations became more frequent, and were more easily reduced. He was treated by massage, exercise, and faradic electricity. In spite of efficient massage, given every other day for several months the arm continued to be dislocated at short intervals and on very slight provocation when the arm was in a position of abduction. Fixation for some weeks with daily massage was advised, but declined, and no treatment was pursued.

CASE III.—A well-built and well-developed man, aged twenty-three years, medical student, consulted Dr. Lovett November 19, 1896. Right arm had been dislocated previously at football; it was fixed for a few

days, and then motion allowed. The dislocation was subglenoid, and was reduced without ether. A little later dislocation was produced by football again, and was reduced without ether. At intervals of a few months dislocations were produced by such causes as reaching up, pitching hay, throwing a stone overhand, and the last dislocation occurred in February, 1896, reaching out over a horse. No dislocation has occurred for over a year, but the patient has carried his right hand in his pocket, and tries not to lift it above the level of his shoulder. At present he is being treated by massage three times a week. The muscular condition has improved and a wider usefulness of the arm has been obtained. Except for the continual and characteristic dread of another dislocation, the patient is but little bothered. The muscles show no change in an electrical examination, and the patient can now reach above his head.

FIG. 2.

FIG. 3.



Case IV.—Showing the atrophy of certain muscles of the left arm.

CASE IV.—Seen by Dr. Lovett February 23, 1897. A professional ball-player. Dislocated his left shoulder October, 1894, in playing foot-

ball, being kicked in the axilla. The dislocation was subcoracoid, and was reduced under ether. The arm was fixed for a few days; two weeks after he began using it, and used it as well as ever.

In April, 1896, he dislocated the arm again in swinging for a standing jump; the dislocation was reduced without ether. Fixation for ten days. The first of February, 1897, he fell on ice, again dislocating the arm—a subglenoid dislocation. He kept the arm quiet for ten days, and three weeks later the arm was still sore.

An x-ray examination showed that the head of the humerus was not as near the acromion as on the other side. It could be restored to place by pressing the arm upward. The capsule was obviously lax, and the humerus "dropped."

The atrophy of the muscles was characteristic. The arm was at once fixed by a sling and swathe for six weeks. Daily massage was given along with faradic stimulation of the atrophied muscles. Then the use of the arm was gradually resumed, and vigorous exercises, both special and general, were given to restore mobility and usefulness. The muscles improved much in appearance and feeling, and by the middle of April he was able to fill his place as catcher of a baseball nine.

CASE V.—Was seen by Dr. Burrell. The patient was twenty-seven years of age, a fisherman by occupation, whose history was as follows:

In August, 1893, fell from a staging and struck on the elbow and shoulder-joint. The dislocation was reduced by a fellow-workman, and

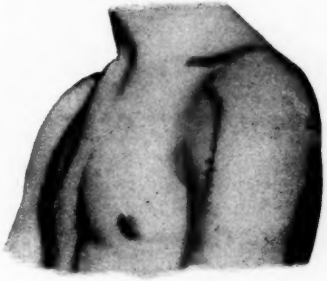
FIG. 4.

Case V.—Before operation, showing the apparent "dropping" of the left humerus.

the patient remained at his work that day. There was no subsequent treatment. There have been numerous recurrent dislocations ever since the accident. At first he used to reduce the dislocation himself and continue work, but of late the joint will not remain reduced for any length of time. Can reduce it more easily than at first. There is some pain in the shoulder.

The patient is well developed and nourished. There is a history of epilepsy. The head of the humerus can be felt apparently in its normal position. There is some atrophy of the deltoid muscle. The sulcus between the deltoid and pectoralis major is very marked, and there is some flattening of the posterior scapular muscles.

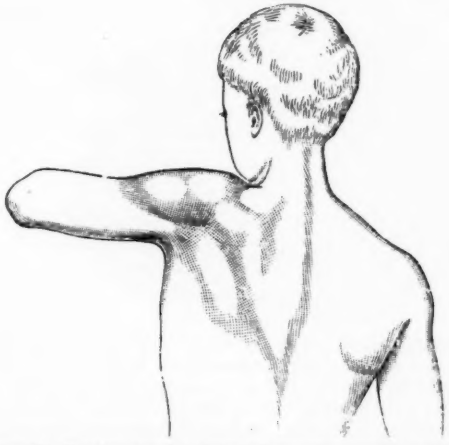
FIG. 5.



Case V.—After operation.

The patient requested that the arm be amputated, as it interfered so seriously with his work. The dislocation has taken place so frequently that he cannot remember the number of times it has occurred. Recently while turning in bed he has frequently dislocated the shoulder-joint.

FIG. 6.



Case V.—Showing atrophy of the left scapular muscles and latissimus dorsi.

On February 19, 1895, an incision was made over the deltoid muscle, beginning at its insertion and extending up about six inches. The fibres of the deltoid were separated, and three-fourths of the tendon of the

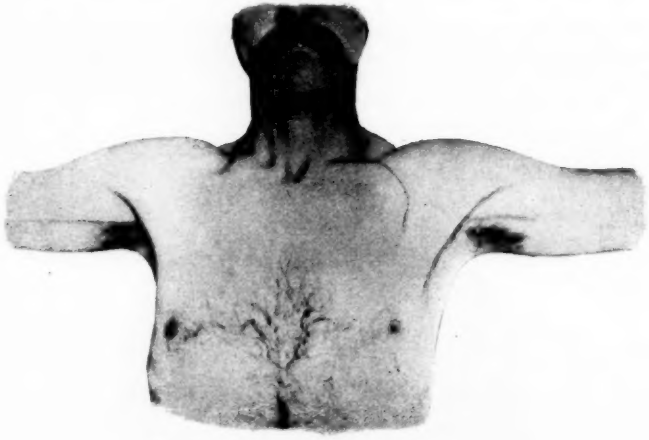
insertion of the pectoralis major was slit down to permit access to the joint. The capsule of the joint was found intact, but very loose. From the inner and anterior aspect of the capsule a piece was removed 4 cm. long and 1 cm. broad. This opened the joint. It was explored, and so far as could be felt was normal.

The capsule was closed by catgut sutures, and the wound was closed by silkworm-gut sutures. A sterile dressing was applied, the arm being put up with the forearm flexed and the point of the elbow raised and carried inward toward the median line.

There was considerable pain after the operation, but it was controlled by morphine. Aseptic healing occurred. The arm was dressed and all the sutures removed in twelve days, but the arm was restrained by a Velpeau bandage. Massage was carried out in this case for four weeks, and the patient returned to his full work at the end of eight weeks from the time of operation.

CASE VI.—Applied at Outpatient Department of the Boston City Hospital in February, 1896, and was operated upon by Dr. Burrell on February 16, 1897. Patient is a well-developed man of medium height, thirty-six years of age, a roofer by trade. During his life has given evidence of "petit mal."

FIG. 7.

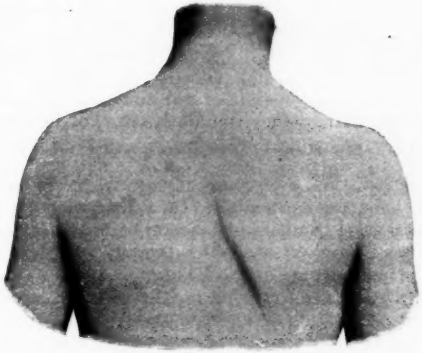


Case VI.—Before operation; front view.

About a year ago, while learning to ride a bicycle he fell off and dislocated his shoulder. It was easily reduced and he continued his ride. Twenty minutes later he ran into a fence and dislocated his arm again. It was again reduced without ether, and was lame for some time, although no surgical treatment was adopted. Since that time he has dislocated the arm thirty-five times. It dislocates with the greatest readiness and from very slight causes. While under observation at the hospital he was asked to raise his hands to the level of his shoulder, which he did, and in lowering them the right humerus slipped and was dislocated into the subcoracoid position; it was easily reduced by the Kocher manipu-

lation. During the past two months he has found that on raising his arm above his head the shoulder would slip out of place. He usually reduces it himself.

FIG. 8.



Case VI.—Before operation, showing the atrophy of the scapular and other muscles of the right side.

FIG. 9.

Case VI.—After operation, showing the cicatrix.

His right shoulder droops. There is a moderate and characteristic atrophy of the muscles about the shoulder-joint. An x-ray examination showed that the tissues over the affected shoulder did not transmit the x-rays as easily as the other. A small amount of dropping of the head of the humerus could be seen. In this second operation a good deal of care was taken to record the various steps in order that it could be presented for publication; *vide* description of operation.

CHOREA IN THE AGED,

WITH THE REPORT OF A CASE OF THE DISEASE IN A MAN AGED SEVENTY-FIVE YEARS.[1]

By David Riesman, M.D.,

ADJUNCT PROFESSOR OF CLINICAL MEDICINE, PHILADELPHIA POLYCLINIC; DEMONSTRATOR OF PATHOLOGICAL HISTOLOGY, UNIVERSITY OF PENNSYLVANIA; NEUROLOGIST TO THE NORTHERN DISPENSARY.

Compared with its frequency in childhood, chorea is so rare in advanced life that the reporting of a new case seems not unwarranted.

The patient, Mr. C. W., a German, is seventy five years of age, married, and an upholsterer by trade. His father died of consumption; the mother and several brothers and sisters of unknown causes. None of his kindred were ever affected like himself, nor does there seem to have been any other neurosis in the family.

He was delicate in his youth and backward in growth, and was for these reasons excused from military service in Germany. He grew five inches in height after the age of twenty-one years. He has always been temperate in the use of tobacco and alcohol, and was seriously ill but once, when he was in hospital one month with some stomach-trouble. He never had rheumatism, but for a short time previous to the onset of his present illness he had pain in his left shoulder. He has always been very constipated, and thinks that he suffers from liver-trouble. For about twenty-five years he has had eczema of the left palm, which at present is worse than ever before. At one time the right hand was similarly affected, but recovered.

The onset of his illness was gradual. He first noticed, in September, 1895, that he could not readily pick up small objects, such as tacks, with the left hand on account of an unsteadiness of movement. The condition increased in intensity until the violent jerkings of the left arm rendered all work impossible. The left leg also began to suffer, becoming unsteady and weak, but it has never presented the extreme inco-ordination affecting the arm. The movements are worse toward evening and after exertion or excitement. They cease entirely during sleep, but seem to delay the oncoming of sleep. The patient's eyesight is good, as is also his memory. There is occasional incontinence of urine, and frequent micturition at night (hypertrophy of the prostate gland). He believes the disease is due to exposure to draughts.

Status præsens. The patient is tall, lank, and pale, but on the whole well preserved for his age. There is well-marked arcus senilis; the pupils react to light and in accommodation, and are equal. The tongue is coated, deflected slightly to the left when protruded, but not tremulous. The radial arteries are thickened and tortuous; the veins of the forearms are unusually prominent and present a well-marked varicose condition. The left hand is the seat of a squamous and fissured eczema covering the entire palmar surface and extending also to the dorsum. On the back of the right wrist is a large purpuric patch, three by one and one-half inches, which appeared spontaneously. The most striking phenomenon presented by the patient is an inco ordinate, grotesque,

[1] Read at a meeting of the Philadelphia Neurological Society, at which the patient was also shown.

tossing movement of the left arm, very wide in its range. The arm is thrown outward and forward and backward; at times it is quickly flexed at the elbow and brought against the trunk. Most frequent is a movement of backward adduction, so that the forearm comes to rest against the posterior part of the left iliac crest. The movements are so violent that they shake the patient's body and often hurl him forward for several feet. They are not constant, but at intervals cease for a few seconds. The left leg is also affected, but only in a slight degree. The gait is sprawling in character, but the left leg is not unduly lifted. That the limb is involved is best seen when the patient attempts to lie down—he then finds that the leg does not obey him, and that he cannot readily bring it up on the couch. After several attempts, in which the limb is tossed about, he suddenly lifts it up high and draws it with a jerk beside the other leg. Unless warned, he always lifts the limb up with his hand.

The will has no direct control over the choreic movements; voluntary motion, as a rule, intensifies them, but at times does not seem to do so. Thus when asked to lift a glass of water to his lips, he does it fairly well, but usually spills it just before drinking. The only way in which he can stop the movements after they have begun is to seize the left forearm with the right hand and hold it in front or behind the chest, the latter being more effectual. This manœuvre also increases the control over the left leg. Voluntary movements of the right arm aggravate the chorea. The grasp is good in the right hand, a trifle weak in the left, but more from interference with proper use than from actual paresis. He can, in fact, not take firm hold with the left hand, as the index-finger tends to remain in a position of over-extension. Speech is somewhat imperfect, but both the man and his wife attribute this to the loss of the teeth; at times, however, the tongue appears a little heavy to the patient. The knee-jerks are absent and cannot be elicited by Jendrassik's method of reinforcement. There is no true ataxia on standing with the eyes closed, although a certain amount of swaying occurs, which, I believe, is ascribable to the choreic movements. Tactile sensation is normal. The patient's mental condition is very good.

There is no notable wasting of the left arm, the difference in size—one and one-half centimetres—found on measuring, is probably due to the fact that the patient is right-handed, and the right arm therefore better developed.

Beyond some accentuation of the second aortic sound, the heart is normal. The temperature is 98° F.; the pulse is 84 in the sitting, 90 in the standing posture. The urine has a specific gravity of 1010, and is free from albumin; the quantity varies from three to five pints in the day.

Résumé. Left-sided hemichorea, involving arm and leg, in a man of seventy-five years; no real impairment of speech; no loss of memory or other psychic defect; movements not controlled by the will; knee-jerks absent; no history of rheumatism; no heart-lesion; duration of disease eight months.

Various types of chorea in adult and advanced life have been described. Herringham,[1] who has studied the different forms, divides them into the following groups:

1. One resembling chorea minor and lasting from a week or two to several months.

[1] Herringham: Brain, April, 1888, p. 134.

2. One due to a coarse subcortical lesion, which follows hemiplegia and sometimes precedes it.

3. The hereditary form of chorea, or Huntington's chorea.

4. An incurable form without hereditary basis and not due to subcortical lesion.

That Sydenham's chorea can occur in the aged has been fully demonstrated. Graves[1] noted it many years ago, and in his clinical lectures refers to two instances. It usually runs a course of from two weeks to several months, and terminates in recovery. Charcot[2] was inclined to consider all choreas of the senile period incurable. I have been able to find in literature thirteen cases ranging in age from forty to eighty-six years in which the disease terminated in recovery. The oldest case ending favorably was one reported by Sinkler—a man aged eighty-six years, who had left-sided hemichorea.

There is, as pointed out by Herringham, not the same tendency to cardiac complications as in juvenile chorea, nor is rheumatism as frequent in the anamnesis. However, in two out of the cases (Carline's and Sinkler's—Nos. 13 and 19 in the table) a heart-murmur existed. The movements are, as a rule, not violent, and the mind is generally not affected; but in the case of Ferrier (No. 17) there was mania, and hebetude in the case of Fry (No 16). In the case of Macleod (No. 34) the patient was maniacal for one month, the duration of the chorea being five months. In other instances the chorea terminated fatally after a more or less brief period of time; at the autopsy the same paucity of lesions was presented as in the chorea of childhood. Grosse (No. 29) noted leptomeningitis. In a case of Charcot's (No. 39) the chorea began at the onset of a lobar pneumonia, from which the patient, a woman, aged sixty-nine years, died. The brain showed diffuse superficial encephalitis. The association of pneumonia and chorea has been observed in childhood. Osler found it in nine out of seventy-three fatal cases of chorea.

Of *Huntington's chorea* but little need be said in this place. The subject has been treated exhaustively by several writers within recent years. The essential features are the onset of choreiform movements in adult life, a progressive dementia, and the existence of the disease in the preceding generation. The affection rarely begins after the age of forty-five. In sixty-six cases tabulated by Huet,[3] the onset was in two instances only after the sixtieth year, and only in ten after the fiftieth year. The movements in Huntington's chorea are very inco-ordinate and more rhythmical than those of Sydenham's chorea, and usually, particularly in the early stages of the disease, are under the control of

[1] Graves: Clinical Lectures. Reprint, 2d edition. Dublin, 1864. In closing his report of one of the cases, he remarks: "Thus do diseases of the nervous system affect a second childhood."

[2] Charcot: Medical Times and Gazette, 1878, 1. p. 245.

[3] Thèse de Paris, 1889.

the will, and are nearly always bilateral. Speech is almost invariably affected, and the hopping gait is quite characteristic.

Posthemiplegic chorea, first described by S. Weir Mitchell and soon after by Charcot, is characterized by choreiform movements of moderate severity, intensified by voluntary effort, and affects especially the distal parts of the extremities, chiefly the fingers and toes. The movements are slow and somewhat athetoid in nature. Hemiplegia is nearly always present, and in some cases is associated with hemianæsthesia; according to Huet,[1] combined motor and sensory paralysis is the most frequent condition. The reflexes are exaggerated on the choreic side.

The lesion giving rise to posthemiplegic chorea is situated subcortically, usually in the neighborhood of (optic thalamus) or within the posterior limb of the internal capsule; exceptionally in the anterior limb. In rare instances, as in the cases recorded by Charcot, Sinkler,[2] Gingeot,[3] and others, the chorea precedes the occurrence of hemiplegia.

There is finally a class of cases, Group 4 of Herringham, which differs from chorea minor in its incurability, and from Huntington's chorea in the absence of the hereditary bias, and in which no gross subcortical lesion exists. A comparatively large number of examples of this variety have been recorded. Whether they in reality belong to a different type and possess their own peculiar pathology may be doubted. Huet[4] maintains that it is not possible to distinguish between chronic chorea with an hereditary basis and chronic chorea without this feature. This view is also taken by Osler.

We have then practically but three forms of chorea in advanced life:

1. *Posthemiplegic* and *prehemiplegic chorea.*
2. *Chorea minor.*
3. *Chronic progressive chorea.* In some of the cases of this type (a comparatively small number after the fiftieth year) there is a distinct history of heredity. The existence of an hereditary tendency seems to induce the development of the disease at an earlier period of adult life, and also predisposes to dementia and other forms of mental failure. In the absence of heredity the onset is late in life, and mental disease is not so apt to occur.

The essential pathological changes are, we may assume, the same in both. In the one type—the so-called Huntington's chorea—the molecular defect of the motor centres and tracts is inherited; in the other it is acquired, as it must originally have been in the individuals who founded the various choreic dynasties. The case here reported seems to belong to the third group—that of chronic progressive chorea.

[1] Huet: Op. cit.

[2] Sinkler: article on Choreiform Affections, in Dercum's Nervous Diseases by American Authors, p. 259.

[3] Gingeot: Gaz. hebdom. de. méd., 1887, 2 S. xxiv. p. 129. Gingeot found nine cases in the literature.

[4] Huet: Op. cit.

No.	Sex.	Age	Supposed cause.	Rheumatism.	Heart.	Parts affected.	Mental faculties.	Duration.	Termination.	Remarks.	Reporter.
1	F.	83	None.	.....	None.	.....	.....	5 weeks	Recovery.	.....	Roger. Gaz. des Hôpitaux, 1854, xxvii. 559. Quoted by Herringham.
2	F.	74	Fatigue from nursing husband	.....	None.	Left side.	.....	11 wks.	Recovery.	.....	Ferguson. Lancet, 1885, ii. 92.
3	M.	70	.....	.....	.....	.....	.....	Many months.	Recovery.	Movements very severe.	Graves. Clin. Lectures. Reprint of 2d ed., p. 406.
4	F.	50	Grief over desertion of husband and children.	.....	.....	.....	Depression of spirits before each attack.	3 years.	.....	Attacks variable; worse on changing of moon.	Graves. Ibid., p. 406.
5	F.	75	Irritation of nerves and shock following extraction of tooth.	.....	None.	.....	.....	2 weeks.	Recovery.	.....	Gauthier. Jour. de Méd. et Chir. Prat., 1881, iii. 66. Quoted by Herringham.
6	M.	68	.....	.....	.....	Right arm and head.	.....	15 yrs.	Death.	Nothing abnormal in brain microscopically.	Herringham. Brain, 1888, p. 134.
7	F.	67	Exposure.	Attack at 24.	Endocarditis of mitral valve.	First, right foot, then the other extremities and the face.	Delusions of persecution.	1 yr. +	Death.	Gyri small and flattened; left lateral ventricle dilated; choroidal cysts; adhesions of spinal pia and dura in cervical region; softening of left cerebellar hemisphere.	Litten. Charité-Annalen, 1886, xi. p. 265.
8	M.	59	.....	.....	.....	.....	.....	.....	.....	.....	Litten. Ibid.
9	F.	60	.....	.....	None.	Right arm and leg.	Normal.	.....	.....	.....	Remak. Neurol. Centralblatt, 1893, xii. p. 538.
10	F.	62	Worry and excitement.	Attack at 45.	None.	Upper and lower extremities, tongue, and lips.	Normal.	9 years.	.....	Had an attack of chorea at 12.	Suckling. Brit. Med. Journ., 1888, i. p. 907.
11	F.	53	Mental anxiety.	None.	None.	Movements general; most marked in left arm and leg.	.....	4 years.	.....	Knee-jerks present; some paresis of left arm and leg. Possibly chorea in mother.	Suckling. Birmingham Med. Rev., 1892, xxxi. p. 209.
12	F.	68	Overwork in a factory.	Attack 2 years before.	.....	.....	.....	6 mos.	Recovery.	Attack severe; non-febrile.	Alexander. Quoted by Mackenzie, Brit. Med. Journ., Feb. 26, 1887.
13	F.	73	Nervous excitement due to drunkenness of husband.	Several attacks; nodules present during attack of chorea.	Aortic systolic murmur.	.....	.....	4 wks.	Recovery.	Attack severe; non-febrile. Several attacks during last three years.	Carline. Quoted by Mackenzie, Brit. Med. Journ., Feb. 26, 1887.

14	F.	78	Diuresis for 7 months, lasting to within 4 mos. of attack.	Chronic articular rheumatism.	Heart weak; no murmur.	.....	.....	40 days.	Death.	Attack severe; febrile. No autopsy.	Bridger. Quoted by Mackenzie, Brit. Med. Journ., Feb. 26, 1887.
15	F.	86	.....	Rheumatic.	.....	.....	Normal.	1 year.	Recovery.	Attack severe.	Aitkin. Quoted by Mackenzie, ibid.
16	F.	69⅔	Poor health.	No distinct attack; slight pains in muscles and joints.	.....	Right side—face, tongue, arm, and leg.	Hebetude	7 wks.	Recovery.	Intercurrent attack of influenza.	Fry. Journ. Nerv. and Ment. Dis., 1891, xviii. p. 596.
17	F.	70	.....	No acute attack; rheumatic pains.	None.	Right side—arm and leg.	Mania.	4 mos.	Recovery.	Previous attack on left side eighteen months before.	Ferrier. Lancet, 1891, i. p. 1379.
18	F.	82	.....	No acute attack; rheumatic pains.	Systolic murmur; irregular heart.	Legs and arm; speech not affected.	Normal.	6 mos.	.....	Movements not severe; controlled by voluntary effort, and then replaced by fine tremor.	Sinkler. Journ. Nerv. and Ment. Dis., July, 1881, p. 577.
19	M.	86	Onset sudden with some loss of power on left side, including face.	.....	Blowing murmur at apex.	Left arm and leg.	Good.	.....	Recovery.	Movements variable in intensity; controlled by the will.	Sinkler. Ibid.
20	F.	48	Blow on head.	None.	None.	.....	.....	.....	Chronic.	Movements came on immediately after blow; knee-jerks exaggerated.	Dercum. International Clinics, 1891, iii. p. 292.
21	M.	58	Bright's disease.	.....	.....	Left side.	.....	.....	Death.	No lesion in brain except œdema and milkiness of membranes.	Dercum. Trans. Coll. Phys. of Philadelphia, 1887, xi. p. 153.
22	M.	60	Bright's disease; a fall.	.....	.....	First, right foot, then leg and arm; later, left side also.	.....	.....	Chronic.	Some loss of power.	Dercum. Ibid.
23	M.	60	Exposure to cold draught.	Acute attack more than 30 years before.	Heart rapid; no murmur.	Neck, back, chest, arms, diaphragm.	Normal.	.....	.....	.....	Anders. Maryland Med. Journ., 1888–'89, xx. p. 467.
24	F.	79	.....	None.	None.	Movements generalized—face, trunk, and limbs.	Impaired.	.....	Death.	Softening of cortex, right side in postero-superior part of ascending parietal, superior parietal, and anterior part of first two frontal convolutions, posterior part of second occipital, also extensive softening on left side. Tissier believes chorea was independent of the softening.	Tissier. Bull. Soc. Anat., Paris, 1889, lxiv. p. 85.
25	M.	58	.....	None.	None.	Lower limbs.	Weak-minded.	4 years.	Chronic.	.....	Nixon. Trans. Royal Acad. Ireland, 1889, vii. p. 58.
26	M.	42	Exposure.	.....	.....	Speech affected.	.....	3 years.	Chronic.	Killed by locomotive.	Hermann. St. Louis Polyclinic, 1889–'90, i. p. 249.
27	M.	54	Worry.	.....	.....	Speech affected.	Impaired.	1 year.	Chronic.	.....	Hermann. Ibid.

No.	Sex.	Age	Supposed cause.	Rheumatism.	Heart.	Parts affected.	Mental faculties.	Duration.	Termination.	Remarks.	Reporter.
28	F.	77	....	None.	None.	Left side chiefly.	Normal.	.....	Recovery.	Attack severe; treatment: zinc sulphate.	Russell. Med Times and Gaz., April 27, 1878.
29	M.	66	.....	.....	Recurrent mitral endocarditis.	Movements at first general, then limited to left side.	.....	3 mos.	Death.	Diffuse chronic leptomeningitis of base and convexity; œdema and hyperæmia of membranes; œdema of brain. Attack of chorea six to eight years before.	Grosse. Berliner klin. Woch., 1889, xxvi. pp. 739, 758.
30	M.	60	.....	.....	Recurrent mitral endocarditis.	.....	.....	.....	Death.	Diffuse leptomeningitis of base and convexity; hyperæmia of brain, attack eight years before.	Koch. Deutsch. Arch. f. klin. Med., 1887, xl. 5, 6, p. 544.
31	M.	52	.....	.....	None.	.....	Good.	12 yrs.	Chronic.	Epilepsy since fifty.	Hoffmann. Virch. Arch., 1888, cxi. p. 532.
32	M.	41	Severe fright.	No rheumatism; several attacks of intermittent fever in childhood.	None.	All four extremities, head, eyelids, tongue; speech slow.	Nearly normal.	1 year.	Chronic.	Knee-jerks normal.	Huet. Thèse de Paris, p. 90.
33	M.	48	Grief on account of infidelity of wife.	Attack at 38	None.	First, right hand, then left hand, face and lower limbs.	Memory impaired.	6 years.	.....	Epilepsy at forty, ceasing after five years; knee-jerks exaggerated.	Huet. Ibid., p. 101.
34	M.	52	No cause; onset gradual.	None.	None.	First, head, then lower limbs, and then upper.	Good.	13 yrs.	Chronic.	Movements violent; knee-jerks exaggerated.	Huet. Ibid., p. 123.
35	F.	49	No cause.	None.	.....	Right side.	Dementia.	2 years.	.....	Epilepsy during first eighteen months of existence of chorea.	Huet. Ibid., p. 140.
36	M.	61	....	No rheumatism; intermittent fever in childhood for 1 year.	None.	Movements generalized, involving face, head, trunk, and extremities.	Memory feeble; intelligence good; ideas of suicide.	Many years.	Chronic.	.....	Huet. Ibid., p. 176.
37	F.	49	.....	.....	.....	Upper extremities, face, head, trunk; less degree lower limbs.	Profound dementia.	8–10 yrs.	.....	.....	Huet. Ibid., p. 181.
38	F.	61	.....	.....	.....	.....	Mania.	4 years.	Death.	Chronic arachnitis; atrophy of brain; excess of fluid in ventricles.	Bacon. Journ. Ment. Sci., 1880, xxvi. p. 253.
39	F.	69	Chorea developed at onset of pneumonia.	.....	None.	Hands.	Much impaired.	Few days.	Death.	Œdema of brain; membranes injected and adherent to anterior and middle lobes, which were red—a diffuse superficial encephalitis.	Charcot. Quoted by Huet, op. cit., p. 229.

40	F.	66	Gradual onset.	None.	.....	Head, face, lips, tongue, trunk; limbs less affected.	Dementia.	15 yrs.	Chronic.	Knee-jerks exaggerated.	Lenoir. Thèse de Lyon, 1888. Quoted by Huet, ibid., p. 256.
41	M.	55	.....	.....	None.	Trunk, limbs, face, tongue, muscles of speech; weakness of lower limbs, especially right.	Impaired.	6 years.	Death.	Pachymeningitis and hæmatoma of dura on both sides; pia congested and adherent; vessels atheromatous; atrophy of convolutions, especially superior parietal lobe; gray matter diminished; ependyma granular; cord soft.	Macleod. Journ. of Ment. Sci., July 4, 1881, p. 194.
42	M.	43	.....	None.	.....	Movements generalized; head also affected.	Diminution of memory.	1 year.	.....	.....	Berdinel. Gaz. Méd. de Paris, 1878, p. 336. Quoted by Huet, op. cit., p. 257.
43	M.	46	None.	None.	None.	All extremities, but chiefly the arms; speech affected.	Impaired.	3 years.	.....	Several previous attacks.	Berdinel, ibid. Quoted by Huet, ibid., p. 258.
44	M.	41	Violent emotion from witnessing killing of a lieutenant.	None.	.....	Right side.	Normal.	6 years.	Chronic.	Onset sudden.	Revillout. Gaz. des Hôp., 1877, p. 801.
45	M.	56	Onset gradual; rapid intensification after death of wife.	None.	None.	Arms, head, and face; to less extent trunk and lower limbs.	Excitable but not demented.	15 yrs.	Chronic.	Knee-jerks exaggerated.	McLearn. Lancet, 1885, i. p. 337.
46	M.	46	.....	.....	.....	Head and arms; speech also affected	Memory impaired, otherwise normal.	.....	.....	Chancre in earlier life.	Mirto. Riforma Med., 1891, vii. 3, p. 289.
47	M.	50	.....	No acute attack.	Aortic valve disease.	Left side, especially arms.	Normal.	.....	.....	Movements to some extent controlled by will.	Saundby. Lancet, 1884, ii. p. 948.
48	M.	87	Exposure to wet.	No acute attack; some enlargement of joints.	None.	Chiefly arms; to less extent head and lower limbs.	No dementia.	5 weeks.	.....	.....	Saundby. Ibid.
49	F.	64	.....	.....	.....	.....	.....	24 yrs.	.....	.....	Bourneville. Le Progrès méd., Aug. 1, 1874, p. 458.
50	M.	68	.....	None.	.....	Arms and head, lips, trunk; lower limbs to less extent.	Good.	18 mos.	.....	.....	Russell. Med. Times and Gaz., 1878, ii. 627.
51	F.	71	.....	.....	.....	.....	Dementia.	12 yrs.	Chronic.	.....	Charcot. Med. Times and Gaz., 1878, i. 245.
52	F.	71	.....	.....	.....	.....	Dementia.	11 yrs.	Chronic.	.....	Charcot. Ibid.
53	M.	40	Rheumatism.	Rheumatic fever at 12 and 40.	.....	.....	.....	.....	Recovery.	A previous attack.	Gowans. Quoted by Mackenzie.
54	F.	40	Blow on back and top of head.	None.	.....	.....	Mania 1 month.	5 mos.	Recovery.	Attack serious; non-febrile.	Macleod. Quoted by Mackenzie.

No.	Sex.	Age	Supposed cause.	Rheumatism.	Heart.	Parts affected.	Mental faculties.	Duration.	Termination.	Remarks.	Reporter.
55	F.	83½	Fall on head 4 years before.	.....	Hæmic murmur at apex.	Movements generalized; limbs, face, head, eye, tongue; right arm and leg were affected; speech affected.	Normal.	3 mos.	Death.	Knee-jerk increased on right side, normal on left side. *Autopsy:* ulcer of clivus Blumenbachii; ecchondrosis of spheno-occipital bone; compression of pons; atheroma.	Muellendorff. Deutsch. Arch. f. klin. Med., 1880, xxvi. 607.
56	F.	80	.....	.....	Heart decrepit.	Movements general; speech affected.	Impaired.	.....	.....	.....	Lloyd. Internat. Med. Mag., 1892, i. p. 593.
57	M.	65	Intemperate; fall on head 20 years before.	.....	.....	Movements generalized; tongue and speech affected.	Mania from intemperance.	6 years.	.....	Had a fit, chorea appearing next day; subsided for a few days, then returned and became violent. Death from acute bronchitis. *Autopsy:* dura thickened, with deposits of lymph on surface, especially base; œdema of membranes; brain pale, sulci deep; atheroma; fluid in auricles.	Hale. Pathol. Trans., London, v. p. 16
58	F.	56	.....	Rheumatism.	None.	.....	Insanity, 5 years.	5 years.	.....	.....	Diller. Amer. Journ. Med. Sci., 1890, xcix. p. 329.
59	F.	59	.....	None.	None.	.....	Impaired.	3½ yrs.	.....	.....	Diller. Ibid.
60	F.	72	.....	.....	Yes.	.....	Insanity, 5 years.	15 yrs.	.....	.....	Diller. Ibid.
61	M.	55	.....	Yes.	None.	.....	Chronic mania.	10 yrs.	.....	Movements lessened since entrance into hospital.	Diller. Ibid.
62	F.	56	.....	Yes.	None.	.....	Chronic melancholia, 5 years.	More than 5 years.	.....	.....	Diller. Ibid.
63	M.	46	.....	None.	None.	Limbs, head, and tongue.	Normal.	6 years.	.....	Improvement under treatment.	Macfaren. Journ. Ment. Sci., xx. p. 97.
64	F.	41	Severe fright at sight of murder.	.....	None; heart irregular.	All voluntary muscles, including those of deglutition.	Weak-minded.	7 years.	Death.	Localized meningitis of membranes in longitudinal fissure, some œdema at base; atrophy of brain; dilatation of vessels, thickening, and amyloid bodies in lymph-sheaths; pigmentary degeneration of many cortical cells; loss of processes, changes in nuclei of 6, 7, 8, 9, 10, 11, 12 nerves; vascular changes in cervical cord; necrobiosis; partial sclerosis of antero-lateral column; inflammatory changes in nerves of brain and cord.	Berkley. Med. News, 1883, xliii. p. 200.
65	M.	75	Exposure to draught.	None.	None.	Left leg and arm; speech not affected.	Normal.	8 mos.	.....	Attack severe; non-febrile; knee-jerks absent.	Personal observation.

I have collected in the appended table the cases of chorea coming on in adult life (after the thirty-fourth year) reported in the accessible literature, but have not included the cases due to gross subcortical lesions. The cases in which a distinct hereditary feature existed have also been omitted, since they have been exhaustively studied from the statistical standpoint by Sinkler, Huet, and others.

The total number of cases tabulated is sixty-five, including the one here reported. Of these, thirty-one (47.7 per cent.) occurred in men, and thirty-four (52.3 per cent.) in women. This proportion is interesting. It differs strikingly from that which obtains in juvenile chorea, as also from that given by other writers on chorea in adults, and corresponds very closely with the incidence of the hereditary form, or Huntington's chorea, in the two sexes. The latter, namely, affects both in nearly equal ratio. The oldest case reported was eighty-seven, the youngest, included in the table, thirty-four years at the inauguration of the disease. Tabulated by decades, the figures are as follows:

Age.	Cases.	Age.	Cases.
30 (34) to 40	6	60 to 70	9
40 to 50	17	70 to 80	8
50 to 60	19	80 to 90	6

From this table it is seen that forty-two (64.6 per cent.) began after the fiftieth year, while of the 66 cases of the hereditary form collected by Huet only 10 had passed that age.

In thirteen out of the sixty-five (20 per cent.) a history of *rheumatism* is given, or articular lesions of a rheumatic nature are described. *Endocarditis* was present in eight cases, or in 12.3 per cent.

In thirteen out of the sixty-five cases (20 per cent.) the chorea was *unilateral*, seven being left-sided and six right-sided hemichorea; but it will be noted that in a number of cases no description of the parts affected is given.

Mental impairment existed in twenty-seven instances (41 per cent.). If the series of five cases reported by Diller from various insane asylums be excluded, the proportion falls to 36.67 per cent. This is in striking contrast with the almost invariable existence of psychic degeneration in the hereditary forms of chorea. *Recovery*, as already stated, occurred in thirteen cases, or in 20 per cent.

The supposed *exciting cause* of the chorea was, in a number of instances, grief or sudden nervous shock; in Gauthier's patient the chorea followed immediately upon the extraction of a tooth In the case here reported exposure to cold draughts is given as the cause.

Conclusions. 1. True Sydenham's chorea may occur in advanced life and may run a short course, terminating in recovery.

2. The majority of cases of chorea occurring late in life run a chronic and progressive course.

3. At the commencement of the disease it is, as a rule, impossible to say whether it will terminate favorably or become chronic.

4. The movements are generally most marked in the arms; they may begin in the lower extremities and, rarely, may remain confined thereto. In 20 per cent. of the cases in the table they were unilateral.

5. The mind is normal in three-fifths of the cases in which no hereditary element exists.

6. Rheumatism holds a very subordinate relation to the chorea of late adult life.

7. Endocarditis is comparatively rare, having occurred in only 12.3 per cent. of the cases collected in the table.

8. The anatomic lesions in the fatal cases are not characteristic.

DISINFECTION BY STEAM.

By Alvah H. Doty, M.D.,
HEALTH OFFICER, PORT OF NEW YORK.

The rapid strides made in bacteriological work during the past ten years have given a stimulus, both in this country and abroad, to the study of disinfection and disinfectants. This has resulted in the accumulation of satisfactory and reliable data, which for the first time place this subject upon a scientific basis. The uncertainty which formerly existed regarding the value of various disinfectants and the manner of their application frequently brought sanitary methods into ridicule. This cannot be wondered at when we examine the long list of disinfectants which have from time to time been confidently recommended, and which we now know to be practically useless as germicidal agents. Fortunately, modern scientific investigation is gradually separating the valuable from the worthless disinfectants. We cannot justly criticise the former conclusions of the sanitarian, as his deductions regarding the value of disinfectants depended almost entirely upon the apparent success which followed their use during the prevalence of infectious diseases. The rapid subsidence of an outbreak was very apt to be attributed in a great measure to the use of certain disinfectants, and an undeserved reputation was frequently given to a worthless article. Today, with a fuller knowledge of the micro-organisms causing the different infectious diseases and the means of studying the effect of different agents upon them, we are enabled to decide with comparative accuracy as to the germicidal power of the latter. Although the experimental stage in the study of this subject may be said to be well advanced, yet the opportunities offered for further investigation in this direction are still great.

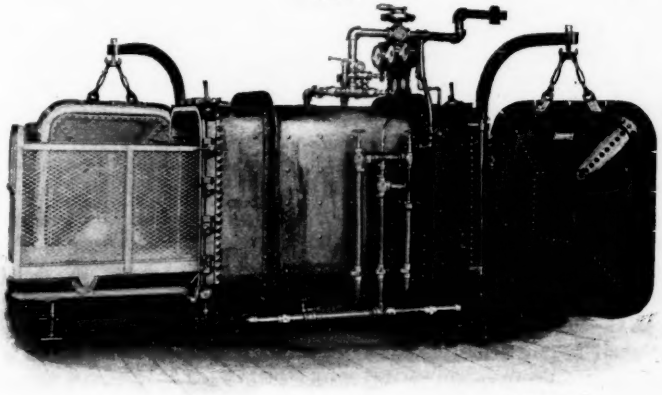
A substance which in the laboratory is germicidal to different micro-organisms with which it is brought in *direct* contact is not always an agent which can be depended upon in practical work. In the latter case the germs are usually not exposed, but are hidden in clothing, bedding, etc. Therefore the penetrating power of a disinfectant constitutes an exceedingly important consideration. Rapidity of action is also an important requisite, particularly in quarantine-work, as is likewise freedom from injurious effects upon delicate fabrics.

Appreciating the necessity of definite and reliable knowledge to guide us in practical work, I have conducted during the past year a series of experiments with some of the disinfectants now in use, and present in this article the results of the work as far as they concern steam.

There can be no diversity of opinion as to the superiority for many purposes of steam over all other disinfectants. The certainty of its germicidal effect, the rapidity of action, and its high penetrating power under proper conditions give it the highest rank. Unfortunately the cost of the apparatus necessary for its proper application limits its use mainly to quarantine and other public service. Although potent when intelligently employed, steam is worthless when improperly applied.

I can, perhaps, give no better idea of the modern perfected steam-chamber than by describing the apparatus for disinfection by steam now in use on the disinfecting-steamer "James W. Wadsworth," at the New York Quarantine Station. (Fig. 1.) This apparatus consists of

FIG. 1.



a rectangular-shaped chamber having a double shell with a space of 2 inches between its walls. The inside dimensions of the chamber are 39 x 48 inches by 7 feet 2 inches long. The shells are made of $\frac{6}{16}$ inch

open-hearth flange steel, riveted at the ends to heavy cast-iron rings, the outer surfaces of which are indented and contain 1½-inch T-shaped rubber joints, for the purpose of making the doors steam-tight. The latter are of a convex type, are swung on cranes, and fastened to the chamber with turn-buckles. A car for the reception of material to be disinfected easily traverses the chamber. It is made of 1½-inch angle-iron and No. 6 galvanized iron netting. A movable track is so arranged that the car can be entirely withdrawn from either end of the chamber. The steam is supplied from the boiler of the steamer, where a pressure of 65 pounds to the square inch is maintained. Both moist and dry steam outlets have been constructed, which, by a system of valves, allow the operator to get almost any quality of steam desired. The steam enters through a 2½-inch pipe to a reducing valve, which limits the pressure of the steam entering the chamber of the apparatus to the desired amount; at the present time it is set for 15 pounds to the square inch. The pressure used in the chamber is in accordance with the temperature required. If a temperature of 230° to 250° F. is desired, steam at about 10 pounds pressure is employed.

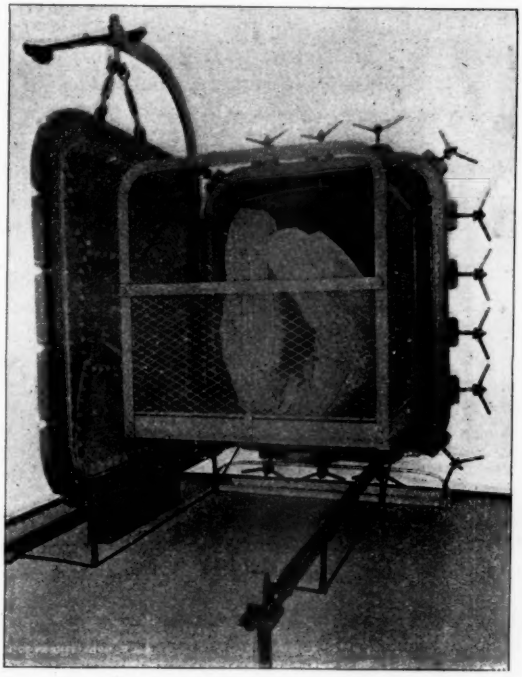
For obtaining a vacuum in the chamber a steam-exhauster is used. By means of valves this is readily changed to draw either from the space between the shells forming the walls, or from the interior of the chamber. It acts on the principle of a siphon: a current of steam rushing by the mouth of a pipe opening into the chamber exhausts the air therein contained.

A vacuum of 1½ atmospheres (about 22 or 23 inches) can be obtained with the steam-exhauster in one minute, while in the old method, by the use of a vacuum-pump, about ten or fifteen minutes were necessary to secure this result; besides, the air removed from the chamber is carried out with the steam in the exhaust, so that if micro-organisms are withdrawn in this way from the materials in the chamber they are acted upon by the steam in transit. This does not occur where a vacuum-pump is used.

The advantages of the double jacket or shell over coils of steam pipes are numerous. There is a more equal diffusion of heat in the walls of the apparatus, inasmuch as the steam enters between the shells at all points and not at the lower part of the apparatus, as in the case where the steam is introduced by pipes laid in the floor of the chamber. The shell is thus evenly heated and materially aids the steam which is introduced in the chamber for disinfection. Leaks are frequently found where the pipe system is employed; these do not occur when the double shell is used; there is no formation of air-bound spaces in the double shell, which frequently interfere with securing the temperature needed; and, finally, in the matter of cleanliness, the double jacket is very superior to the coils of pipes.

The fresh-air inlet, which, as far as I am able to learn, was first used at this Station, and which has proved of great practical value, consists of a 1½-inch pipe connecting the interior of the chamber with the external air. It is fitted to the top of the apparatus. The external opening of the inlet is covered with a strainer of fine brass netting, to prevent the entrance of foreign matter. The inlet is supplied with a valve, to open and close it. The entire chamber is encased in a 1½-inch layer of asbestos magnesia, which retains the heat in the chamber and prevents

FIG. 2.

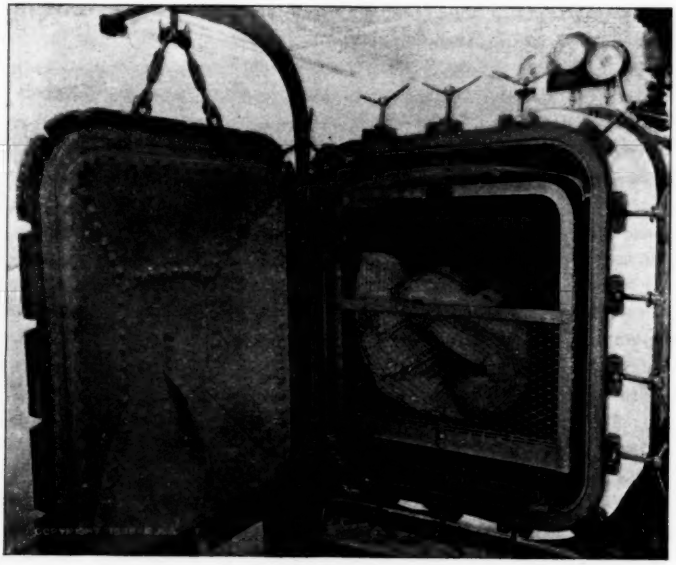


Infected end.

outside radiation. This it does in a very effective manner, the temperature of that portion of the boat in which the apparatus is placed being very little raised above that of adjoining apartments. The ends of the chamber open into rooms having absolutely no communication with each other, the infected end (Fig. 2) receiving the clothing removed from the disrobing-room adjoining. The disinfected end

(Fig. 3) opens into an apartment from which the clothing is given directly to the owners, who have passed through the bath-rooms. The operation of disinfecting clothing, bedding, etc., with the steam-chamber is as follows: the materials to be treated are placed in the cradle or car in the infected end, which is seen in Fig. 2 (both ends of the chamber are never open at the same time), the door is then bolted, and a vacuum of about 20 inches is obtained by the steam-exhauster. This is secured in about one minute. The object is not only to remove the air from the

FIG. 3.



Disinfected end.

chamber, but also the air and moisture from the material to be treated, which, forming a cushion, prevent the proper entrance of the steam. Steam is now allowed to enter the chamber, which it does very rapidly, producing a temperature of 230° to 240° F. within three or four minutes. My experience has been that 230° is the temperature that is needed for disinfection. This is unquestionably safe, and can be depended upon to kill all known micro-organisms which may be in clothing and other articles presented for treatment, provided there is an exposure of fifteen minutes to this degree of heat. At the expiration of this time—*i. e.*, after an exposure of fifteen minutes to a temperature of 230°—the steam-exhauster is again used, securing the same degree of vacuum (20

inches). This now removes the steam from the chamber and from the texture of the clothing, etc, undergoing disinfection, the latter being a most important consideration, as will be shortly shown. After the vacuum of 20 inches has been obtained the fresh-air inlet is opened and a current of fresh air allowed to pass through the chamber and its contents. This, it must be understood, is in conjunction with the steam-exhauster, which is still kept at work. The entrance of the air, of course, breaks the vacuum, but the exhauster carries the air rapidly through the chamber. After an exposure of eight or ten minutes to this treatment the door in the disinfected end is opened, and the clothing, etc., after being unrolled and exposed for five or six minutes, becomes dry and can be at once worn.

The degree of improvement in the drying-process, which is the result of the combined action of the steam-exhauster and the fresh-air inlet, can only be appreciated by one who has waited two or three hours or more for this same result. When it involves the detention of a ship at a quarantine-station its value is at once apparent. The importance of the rapidity with which this work is now performed and the materials dried is especially appreciated in the treatment of mail. Letters and papers treated with steam by the slower or older process were frequently injured by the disfigurement of the address and the unsealing of the letters. During the past three months the British India mail has been disinfected weekly at this station. The few letters which are sealed with wax, and packages containing books or other materials injured by heat, are set apart for disinfection by other methods and the remainder subjected to the process indicated above. So far, I have yet to see a letter the superscription of which has been affected, and very rarely have I found a letter unsealed or partly unsealed. This satisfactory result, I am certain, is due to the rapidity of action of the heat, and the use of the exhauster and fresh-air inlet afterward, which rapidly dries and prevents the spreading of the ink and the unsealing of the package.

The still more important and valuable effect of the vacuum is shown in the rapid and pronounced penetration which it insures. It gives a higher degree of temperature inside the packages, with a comparatively short exposure.

The first series of tests presented for consideration show the influence of the vacuum in steam disinfection. In all the experiments which I have made I constantly used packages of paper, inasmuch as this material probably offers as much (if not more) opposition to penetration as any with which we have to deal. In the tests made without vacuum, although the steam-exhauster is not used, the outlet which leads from the bottom of the apparatus and which allows the escape of water, the result of condensation, is opened for a moment after the chamber is closed, to allow a certain amount of the air contained in

the chamber to be forced out by the incoming steam. This vent is imperative in carrying out the disinfection, as in an absolutely tight chamber the presence of all the confined air offers so much resistance that it is practically impossible to raise the temperature to the proper degree for disinfection. When the chamber is perfectly closed it frequently requires half an hour or more to get a temperature of 210° F.; whereas when the steam-exhauster is used 240° F. can be reached in three or four minutes, and where the air in the chamber is simply released by opening the conductor on the floor of the chamber above referred to, the same temperature (240° F.) is attained in about eight or ten minutes. This, however, does not answer the same purpose as the vacuum, which forcibly draws out the air contained in the chamber and its contents.

In the experiments about to be considered an accurately tested self-registering thermometer was placed in the centre of each package enumerated in the different experiments.

TABLE I.

Article.	Weight.	Subjected to degree of temperature.	Time of exposure.	Thermometer in package registered	
				With vacuum.	Without vacuum.
	Lbs.	°	Min.	°	°
Newspaper	1	240	3	240	200
Newspaper	2	240	3	210	160
Newspaper	2	240	3	210	160
Newspaper	3	240	3	200	110
Newspaper	3	240	3	200	111
Newspaper	3	240	3	220	120 (loosely wrapped)
1 cotton sheet	1	240	3	240	240
2 cotton sheets	2	240	3	239	237
1 double rose blanket	4	240	3	239	185
2 double rose blankets	8	240	3	203	110
1 ordinary house-rug	7	240	3	238	235
1 bag woollen clothing	7	240	3	173	108

TABLE II.

Article.	Weight.	Subjected to degree of temperature.	Time of exposure.	Thermometer in package registered	
				With vacuum.	Without vacuum.
	Lbs.	°	Min.	°	°
Newspaper	1	234	10	230	218
Newspaper	2	234	10	212	200
Newspaper	3	234	10	190	170
2 double blankets	8	234	10	215	190
2 sheets	2	234	10	230	229
1 bag woollen clothing	7	234	10	230	232

TABLE III.

Article.	Weight.	Subjected to degree of temperature.	Time of exposure.	Thermometer inside package registered
With vacuum:	Lbs.	°	Min.	°
Newspaper	1	240	10	240
Newspaper	2	240	10	232
Newspaper	3	240	10	200
2 cotton sheets	2	240	10	240
2 double rose blankets	8	240	10	188
1 bag woollen clothing	7	240	10	230
Without vacuum:				
Newspaper	1	244	10	240
Newspaper	2	244	10	210
Newspaper	3	244	10	196
2 cotton sheets	2	244	10	235
2 double rose blankets	8	244	10	195
1 bag woollen clothing	7	244	10	205

TABLE IV.

Article.	Weight.	Subjected to degree of temperature.	Time of exposure.	Thermometer inside package registered
With vacuum:	Lbs.	°	Min.	°
Newspaper	2	230	10	215
Newspaper	2	230	10	195
Newspaper	3	230	10	170
Without vacuum:				
Newspaper	2	227	10	133
Newspaper	2	227	10	174
Newspaper	3	227	10	120

It will be seen that the effect of the vacuum in favoring penetration is more apparent in short exposures. Where the exposure is longer the difference in the temperature, as shown by the thermometers inside the packages, is not so great. This is shown in Table I. In this table it is shown that the temperature inside a package of paper weighing two pounds was 210° F. after an exposure of three minutes with vacuum; while the temperature inside a package of the same weight without vacuum was only 160° F., a difference of 50° F.; whereas in Table II., after an exposure of ten minutes the difference in temperature inside of a package of paper weighing two pounds, with and without a vacuum, was only 120° F. It will be noticed that the effect of the vacuum is more decided in the three-pound packages of paper, or in any material where the resistance to the penetration of steam or heat is pronounced. This is also shown in Tables I. and II., where the thermometers were wrapped in cotton sheets, which are very easily penetrated; the temperature was about the same with and without vacuum.

In Table I. a bag of woollen clothing showed a temperature inside the package of 172° F. with vacuum and 108° F., without vacuum; while

in Table II. the temperature inside the bag of clothing was practically the same with and without vacuum, and about equal to the temperature of the chamber. In Table III. there is a marked difference between the test with a bag of clothing made with and without vacuum. These discrepancies are easily explained in this particular case: the clothing was simply pushed into the bag after the manner of sailors, and after each test the clothing was removed and aired again and placed in the bag for the succeeding test, and was not packed with the same degree of tightness in each instance, nor could the thermometer always be protected to the same extent. However, the advantage of the vacuum is shown in two of the three tests. In Table IV., where the experiments were made with packages of newspapers, the advantage of the vacuum is again evident.

It was meant that the temperature of the chamber should be the same in each instance where the tests with and without vacuum were to be compared; but occasionally the steam-pressure becomes somewhat unruly, and a difference of a few degrees is noticeable, as in Tables III. and IV. In these cases it was necessary to divide the tables. However, a fair comparison can be made.

It is frequently observed that the temperatures registered in packages of the same material and weight and subjected to the same temperature in different tests do not always agree. Thus, a package of newspapers weighing two pounds and subjected to a temperature of 240° F. for ten minutes may show an inside temperature of 230° or 232° F., while the temperature in the same test at another time will show a difference of 10° or more. This is due to the way of folding the package, which cannot be performed in exactly the same manner in each instance. If the material is more tightly wrapped, the temperature inside is correspondingly less. This occurs where the greatest care is exercised.

The results detailed in the preceding tables also indicate the importance of a sufficient duration of exposure. In Table I. it is shown that the temperature within a package of newspapers weighing two pounds, and subjected to a temperature of 240° F. for three minutes, was 210° F. with vacuum, whereas after an exposure of ten minutes to the same temperature it was 232° F. In the above experiments the steam-pressure in the chamber necessary to secure the temperature required (about 230° F.) was from eight to twelve pounds to the square inch. In the tests made with a perfectly tight chamber, with no release of the confined air, it was practically impossible to obtain a sufficiently high degree of heat. Even a comparatively low temperature required from half to one hour. In one instance, with a pressure of fifteen pounds to the square inch, a temperature of 202° F. only was reached in forty-five minutes; whereas, as already shown with the steam-exhauster, a temperature of 240° F. was reached in three minutes. In another test,

without any vent in the chamber, 215° F. was reached in about the same length of time (less than an hour). I believe, therefore, it is unnecessary to consider further the process of disinfection as performed in a perfectly tight chamber.

In the investigation of steam disinfection it soon becomes apparent that as the degree of pressure in the chamber diminishes the penetration is correspondingly less. The following tables, V. and VI., will illustrate this. In these tests there was no steam-pressure in the chamber whatever, the indicator registering nothing. It will be seen by referring to Table I. that in the package containing two cotton sheets penetration was very easily effected, the thermometer inside the package being within four degrees of the temperature of the chamber (240° F.) after an exposure of only three minutes, while Table V. shows a difference of 48° F. between the temperature of the chamber (170° F.) and the temperature inside the package of sheets after an exposure of ten minutes. This marked difference is also seen in Table VI.

TABLE V.

Article.	Weight.	Subjected to degree of temperature.	Time of exposure.	Thermometer inside package registered
With vacuum:	Lbs.	°	Min.	°
Ordinary house-rug	7	170	10	100
Newspaper	1	170	10	137
Newspaper	2	170	10	120
Newspaper	3	170	10	107
2 double rose blankets	8	170	10	120
2 sheets	2	170	10	122

TABLE VI.

Article.	Weight.	Subjected to degree of temperature.	Time of exposure.	Thermometer inside package registered
Without vacuum:	Lbs.	°	Min.	°
1 rug	7	200	10	105
Newspaper	1	200	10	160
Newspaper	2	200	10	120
Newspaper	3	200	10	100
2 double rose blankets	8	200	10	135
2 sheets	2	200	10	165

The results given in the above tables have been repeatedly verified by numerous tests of the same character.

These preliminary tests were regarded as necessary to the proper investigation of the effects of steam under the given conditions on the bacillus of the bubonic plague and the diphtheria and anthrax bacilli, the results of which are given below. In order that there should be no question as to the vitality of the micro-organisms used, the cultures were

freshly prepared in each test by Dr. C. B. Fitzpatrick, who carefully maintained their virulence by inoculation of guinea-pigs and white mice. This procedure was carried on in connection with his work with the plague antitoxin, a report of which has recently been published in the *New York Medical Journal.* Sterilized linen disks soaked in bouillon cultures and enclosed in cotton, woollen, and paper envelopes, were placed inside the packages used in the different experiments.

It was found in all cases, where linen disks soaked in cultures of the bacilli of the above-mentioned diseases were placed in open Petri dishes and then laid on the floor of the cradle in the steam-chamber, and subjected to a temperature of 155° F. for fifteen minutes, that no growth occurred. I believe that it is safe to regard this temperature as causing the death of the bacilli in question in all cases under these conditions, although the germs were usually killed at a temperature of about 135° F. It is to be understood, however, that a temperature of 155° F. must reach the interior of the package. A chamber-temperature of this degree (155° F.) would probably show not more than 100° F. inside the package, and would be manifestly insufficient even with a long exposure. By referring to Tables V. and VI. it will be seen that in but two instances did the temperature inside the packages reach 155° F., and this occurred once in a package of one pound of newspaper and once in one composed of two sheets, where an exposure of ten minutes to a temperature of 200° F. was made. The packages were removed from the chamber in each experiment immediately after the exposure and the disks contained therein placed in tubes containing bouillon. The tubes were kept in the incubator for at least a week before a final decision as to the growth of the bacilli was made.

The facts secured in the first series of experiments (with thermometers) relative to penetration, etc., corroborate the tests made in the second series (with micro-organisms), and show that where the packages were subjected to low temperature growths frequently occurred; and although a lower temperature may cause the death of the micro-organisms subjected to it, it is unsafe to compute too closely the degree of heat necessary for germicidal action; therefore considerable margin should be allowed. For this reason I believe that a temperature of at least 155° F. must be secured inside the packages to be disinfected. The tests described above made with self-registering thermometers in the first series of experiments may be used as a guide. A study of the following tables shows that where packages containing infected disks were subjected to comparatively low temperature the results were variable. This is well shown in Tables VII. and VIII. (second series), where packages of the same material and weight were subjected to the same temperature and exposure (170° F. for ten minutes).

Second Series.

Table VII.—With Vacuum.

Temperature, 170° F. Time of exposure, 10 minutes.

Plague:	
1. Sheet of paper and envelope	No growth.
2. Newspaper, 1 pound	Growth.
3. Newspaper, 2 pounds	Growth.
4. Blanket	No growth.
Diphtheria:	
1. Sheet of paper and envelope	No growth.
2. Newspaper, 1 pound	No growth.
3. Newspaper, 2 pounds	No growth.
4. Blanket	No growth.
Anthrax:	
1. Sheet of paper and envelope	No growth.
2. Newspaper, 1 pound	No growth.
3. Newspaper, 2 pounds	No growth.
4. Blanket	No growth.

Table VIII.—With Vacuum.

Temperature, 170° F. Time of exposure, 10 minutes.

Plague:	
1. Sheet of paper and envelope	No growth.
2. Newspaper, 1 pound	Growth.
3. Newspaper, 2 pounds	Growth.
4. Blanket	Growth.
Diphtheria:	
1. Sheet of paper and envelope	No growth.
2. Newspaper, 1 pound	No growth.
3. Newspaper, 2 pounds	Growth.
4. Blanket	No growth.
Anthrax:	
1. Sheet of paper and envelope	No growth.
2. Newspaper, 1 pound	Growth.
3. Newspaper, 2 pounds	Growth.
4. Blanket	Growth.

Nothing can better illustrate the unreliability of a low temperature in steam-disinfection than the experiments shown in Tables VII. and VIII. The infected disks enclosed in paper and envelope show "no growth" in all the tests; beyond this there is no uniformity in the result, even where the disks were in a single newspaper. A reference to Tables V. and VI., where packages of similar material and weight

(as used in Tables VII. and VIII.), and containing self-registering thermometers, were subjected to the same temperature and exposure, will present a satisfactory explanation of the irregular results shown in Tables VII. and VIII. It will be seen in the experiments in Tables V. and VI. that an inside temperature of 155° F. (which may be regarded as the minimum necessary to insure destruction of the bacilli) was reached in only two instances, both in Table VI., and, as might be expected, in material which practically offered the least resistance. (See Table VI., one pound of newspaper and two sheets.)

TABLE IX.—WITH VACUUM.

Temperature, 200° F. Time of exposure, 10 minutes.

Plague:	
1. Sheet of paper and envelope	No growth.
2. Newspaper	No growth.
3. Newspaper, 1 pound	Growth.
4. Newspaper, 2 pounds	Growth.
5. Blanket	No growth.
Diphtheria:	
1. Sheet of paper and envelope	No growth.
2. Newspaper	No growth.
3. Newspaper, 1 pound	No growth.
4. Newspaper, 2 pounds	Growth.
5. Blanket	No growth.
Anthrax:	
1. Sheet of paper and envelope	No growth.
2. Newspaper	Growth.
3. Newspaper, 1 pound	[1]
4. Newspaper, 2 pounds	Growth.
5. Blanket	Growth.

It is of great interest to note that in Table IX., where the temperature of the chamber in the test was increased to 200° F. (30° more than in the experiments recorded in Tables VII. and VIII.), that the germicidal action was more apparent. This is also shown in the following Tables, X., XI., and XII.

TABLE X.—WITH VACUUM.

Temperature, 200° F. Time of exposure, 15 minutes.

Diphtheria:	
1. Blanket	No growth.
2. Newspaper	No growth.
3. Newspaper, 1 pound	Growth.
4. Newspaper, 2 pounds	Growth.
5. Magazine, 200 pages, disks in middle near binding	Growth.

[1] Tube of bouillon containing infected disk broken.

TABLE XI.—WITH VACUUM.

Temperature, 202° F. Time of exposure, 5 minutes.

Plague:	
1. Sheet of paper and envelope	No growth.
2. Newspaper, 1 pound	No growth.
3. Newspaper, 2 pounds	Growth.
4. Magazine, 160 pages, disks in middle near binding	No growth.

TABLE XII.—WITH VACUUM.

Temperature, 202° F. Time of exposure, 10 minutes.

Plague:	
1. Sheet of paper and envelope	No growth.
2. Newspaper, 1 pound	No growth.
3. Newspaper, 2 pounds	Growth.
4. Magazine, 160 pages, disks in middle near binding	No growth.
5. Blanket	Growth.

In Table XI., with an exposure of five minutes to a temperature of 202° F., the disks placed in the rolled blanket showed no growth; while in Table XII., with an exposure of double the time (ten minutes), the disks in a similar package (blanket) show a growth. This undoubtedly is the result of a difference in the degree of tightness with which the blanket was wrapped, and is often noticed where the test-articles have been exposed to comparatively low temperature; whereas with higher temperature given with shorter exposure no way in which a blanket or other package is rolled can interfere sufficiently with the penetration to prevent germicidal action. This is well illustrated in the experiments which follow, showing tests made with higher temperature.

In the following table the temperature of the steam-chamber was raised to 215° F., an increase of about 10° over that used in the experiments cited in Tables X., XI., and XII. For the first time we find the bacilli killed in all the packages.

TABLE XIII.—WITH VACUUM.

Temperature, 215° F. Time of exposure, 10 minutes.

Plague:	
1. Sheet of paper and envelope	No growth.
2. Book, 600 pages	No growth.
3. Newspaper, 1 pound	No growth.
4. Newspaper, 2 pounds	No growth.
5. Blanket	No growth.
Diphtheria:	
1. Sheet of paper and envelope	No growth.
2. Book, 600 pages	No growth.
3. Newspaper, 1 pound	No growth.
4. Newspaper, 2 pounds	No growth.
5. Blanket	No growth.

The anthrax-tests in this group of experiments are omitted, as unfortunately the labels of some of them were partially obliterated and made uncertain.

The following Tables, XIV., XV., XVI., XVII., XVIII., and XIX., are severe tests, inasmuch as they include packages of paper weighing three pounds. I do not believe that any material or bundle which might be presented for disinfection would be able to resist the penetration of steam to such an extent as some of the packages enumerated in the following tables, and I am firmly convinced from the experimental work and results gained in the foregoing experiments that a temperature of 230° F. in a steam-chamber, with an exposure of fifteen minutes, and the use of a vacuum will kill whatever vegetative organisms (and also probably spores) there may be in the material presented for disinfection, either at quarantine-stations or in public institutions. It is understood, however, that there should be a proper apparatus and that the work should be carefully done by a competent operator.

TABLE XIV.—WITH VACUUM.

Temperature, 230° F. Time of exposure, 5 minutes.

Plague:	
1. Newspaper, 16 pages	No growth.
2. Newspaper, 1 pound	No growth.
3. Newspaper, 2 pounds	No growth
4. Book, 600 pages	No growth.
5. Blanket	No growth.
Diphtheria:	
1. Newspaper	No growth.
2. Newspaper, 1 pound	No growth.
3. Newspaper, 2 pounds	No growth.
4. Book, 175 pages	No growth.
5. Blanket	No growth.
Anthrax:	
1. Newspaper, 16 pages	No growth.
2 Newspaper, 1 pound	No growth.
3. Newspaper, 2 pounds	No growth.
4. Book, 600 pages	No growth.
5. Blanket	No growth.

TABLE XV.—WITH VACUUM.

Temperature, 230° F. Time of exposure, 5 minutes.

Plague:	
1. Sheet of paper and envelope	No growth.
2. Book, 600 pages	No growth.
3. Newspaper, 1 pound	No growth.
4. Newspaper, 2 pounds	No growth.
5. Blanket	No growth.

Diphtheria:

1. Sheet of paper and envelope . . . . No growth.
2. Book, 250 pages . . . . . No growth.
3. Newspaper, 1 pound . . . . . No growth.
4. Newspaper, 2 pounds . . . . . No growth.
5. Blanket . . . . . No growth.

TABLE XVI.—WITH VACUUM.

Temperature, 230° F. Time of exposure, 10 minutes.

Plague:

1. Disks in book, 600 pages, book tied . . . No growth.
2. Disks in single blanket . . . . . No growth.
3. Disks in package of newspapers, 3 pounds . No growth.

TABLE XVII.—WITH VACUUM.

Temperature, 230° F. Time of exposure, 10 minutes.

Plague:

1. Almanac, 30 pages . . . . . No growth.
2. Magazine, 15 pages . . . . . No growth.
3. Magazine, 150 pages . . . . . No growth.
4. Book, 600 pages . . . . . No growth.
5. Newspaper, 2 pounds . . . . . No growth.

TABLE XVIII.—WITH VACUUM.

Temperature, 230° F. Time of exposure, 15 minutes.

Plague:

1. Sheet of paper and envelope . . . . No growth.
2. Sheet of paper and envelope . . . . No growth.
3. Newspaper, 1 pound . . . . . No growth.
4. Newspaper, 2 pounds . . . . . No growth.
5. Mattress (Mexican moss) . . . . . No growth.
6. Mattress (excelsior) . . . . . No growth.
7. Blanket . . . . . No growth.
8. Coats with disks in pockets . . . . No growth.

TABLE XIX.—WITH VACUUM.

Temperature, 230° F. Time of exposure, 15 minutes.

Plague:

1. Newspaper, 2 pounds . . . . . No growth.
2. Newspaper, 3 pounds . . . . . No growth.
3. Blanket . . . . . No growth.

Diphtheria:

1. Newspaper, 2 pounds . . . . . No growth.
2. Newspaper, 3 pounds . . . . . No growth.
3. Blanket . . . . . No growth.

Anthrax:

1. Newspaper, 2 pounds . . . . . No growth.
2. Newspaper, 3 pounds . . . . . No growth.
3. Blanket . . . . . No growth.

Experiments similar to the above have shown that a temperature of 230° F., with an exposure of fifteen minutes, is also destructive to the spores of anthrax.

In conclusion, I would reiterate what I have already said, that in order to procure the desired results in steam disinfection careful attention should be given to the selection of the steam-chamber; that a temperature of 230° F. and an exposure of fifteen minutes are required; and that for quick and effective work a steam-exhauster should be provided. These requirements are particularly needed at quarantine and other public disinfecting-stations. At these places it is entirely impracticable to open packages and spread them out for exposure, consequently the matter of penetration must always be carefully considered. It will be seen in the tests made with low temperatures that the pressure in the chamber was practically zero, and, as might be inferred, the penetration was ineffective. If this is the result in a tight chamber, it cannot be expected that any safe or satisfactory result can follow where steam is indiscriminately introduced into a living-apartment or in a dwelling or on board a vessel. This I know has from time to time been advocated. I do not believe that a living-apartment or hold of a ship can be made sufficiently tight or can be subjected to a steam-pressure great enough to disinfect its contents properly. Leaks are sure to occur, which tend at once to render the steam ineffective as a germicide. If it should be desired to disinfect the hold of a ship, and steam should be selected as the agent, it is apparent that a temperature applied to the inner surface of the hull of the vessel below 155° F. would be unsafe to depend upon. There is no question that the organisms which would be found in an infected hold would, in part, rest upon the inner sides of the vessel. Wooden ships are becoming a thing of the past, and iron ones are being substituted. These rest in water the temperature of which is always low, and a superficial consideration will show that as the result of conduction the hull is always cold. I do not believe the low temperature of the hull can possibly be raised to a sufficient degree by the amount of steam which can be thrown into this part of the vessel, taking into consideration the numerous leaks which are always present. Fortunately, the ship's hold and cargo are rarely infected, and are too frequently subjected to a worthless disinfection.

REVIEWS.

TRAITÉ DE MÉDECINE ET DE THÉRAPEUTIQUE. Publié sous la direction de MM. P. BROUARDEL, Membre de l'Institut, Doyen de la Faculté de Médecine de Paris, Médecin de la Charité; A. GILBERT, Professeur agrégé à la Faculté de Médecine de Paris, Médecin de l'hôpital Broussais; et J. GIRODE, Médecin des hôpitaux de Paris, Auditeur au comité d'hygiène publique de France. Tome premier, Maladies Microbiennes. Pp. vii., 818. Paris: J. B. Baillière et Fils, 1895.

TREATISE ON MEDICINE AND THERAPEUTICS. By BROUARDEL, GILBERT, and GIRODE. Microbial Diseases.

To adequately review this, the first of the series of ten volumes, would require far more space than can be spared in the press of important matter which so constantly crowds our pages. Therefore we must content ourselves in calling attention to the salient points of this, an epoch-maker among the Systems of Medicine. At the outset, this volume —devoted to a discussion of the diseases of parasitic origin—stands unique in that not only the best and most carefuly reached results of laboratory study are presented, but equally the conclusions of practised clinicians arrived at from painstaking analysis. It is but a few years ago that bacteriological work might have been truly said to have been barren of therapeutic suggestion. Today the practitioner, be he physician or surgeon, must keep himself in touch with the latest and best from the laboratory if he strives for his highest success. The Introduction, Microbian Diseases in General, by M. J. Girode, is brilliant. To one who has followed the subject, as the current literature affords examples, the extent and the intimate relationship of this subject with that of practical work come as a revelation. We venture to predict that upon this chapter many papers, yet to be written, will be based. Of the special subjects, Variola, by M. B. Auchè, bears testimony to the value of preventive measures, but does not pass upon the most recent but not yet successfully demonstrated exhibitions of serumtherapy. M. H. Surmont takes up Vaccine, covering substantially the same ground which has been so recently traversed in the literature brought out by the anniversary of Jenner, and in it the antivaccinationists can find but little comfort, for all that he spares these a recital of the bitter lessons taught at Gloucester. M. L. Galliard considers Varicella, denying its relationship with vaccinia, varioloid, and variola, although admitting that this opinion may be modified by future bacteriological experiments. A discreet and carefully prepared section is that upon Scarlet Fever, by M. R. Wurtz, although the treatment should have received more attention. The generally neglected subject of Measles is thoroughly and ably presented by M. J. Grancher, while

M. M. Netter devotes a brief chapter to so-called German Measles, especial attention being given to its non-identity and differentiation from the former. Miliary Fever, of interest chiefly to the well-informed physician, has been intrusted to M. L. Thoinot. The extensively written-about epidemic Influenza receives careful treatment by M. A. Netter, who assigns Pfeiffer's bacillus (1892) as the causative agent. His treatment is symptomatic, and he warns against the use of antipyretics in large doses; we could wish that upon this point he had been more emphatic. The same author takes up Dengue, as it seems, in a somewhat perfunctory manner. We are of the opinion that the disease must be observed or experienced to be properly appreciated. Whooping-cough and Mumps are both discussed by MM. A. Legroux and L. Hudelo, who are rather verbose and offer, beyond an excellent summary of existing knowledge, but little of novelty. For Diphtheria we have M. J. Grancher, who is here assisted by M. P. Boullache. We read this chapter with interest, for here therapeutics has received most signal assistance from the laboratory. This is short but satisfactory, temperate in tone, and our only regret is that the more recent developments could not have found a place and made more emphatic their conclusions, which were reached in November, 1894. M. F. Widal writes upon Streptococcal Disease and Facial Erysipelas in a masterly manner, omitting only the work of Marmorek. M. J. Courmout gives an excellent presentation of the *rôle* of the staphylococcus in pathology (Staphylococcal Diseases). Why may we not now transfer *staphylococcie* to our language? As our disease-concepts become more exact, our nomenclature must be more precise. An exceedingly tersely written chapter is that by M. L. Landouzy on Pneumococcal Disease. Here the pneumococcus is studied apart from acute fibrinous lobar pneumonia. Following Foa, the micro-organism may be (1) the pneumococcus proper, œdematogenous acting by its toxins, or (2) the meningococcus, fibrinogenous acting by sepsis. The relationship of the diseases formerly considered to be distinct was suggested, on clinical grounds only, by us in 1887. The hope in the direction of therapy evidently lies in immunization. M. A. Gilbert considers the *bacterium coli commune* in its distribution, physiological and pathogenic *rôle* (colibacillosis), and closes by stating that this may be a useful germ or a vigilant enemy. Thus far have we read this volume with great satisfaction; it presents the various subjects with but little departure from the high standard set in the Introduction. Ordinarily, when a conjoint work receives the criticism that its sections are of unequal value, the real significance is that some are excellent, others good, and some hopelessly bad. In this volume we find none unworthy of the subject or of the author. Yet excellent as it is, the final chapter upon Enteric Fever, by MM. P. Brouardel and L. Thoinot, is most remarkable for its thoroughness, for its logical deductions, for its scholarly presentation, and for its scientific breadth. We well remember the Brouardel of our student-days—clear-headed, earnest, and learned—but much as we have expected, here we rest satisfied. In its completeness we note only the omission of so-called myoidema, which is frequently found in the later periods of the disease. We would especially call attention to the summary: (1) combined use of antipyretics, cold lotions, or wet-sheet and tonics, mortality 11.97 per cent.; (2) quinine and tepid baths, 7.33 per cent.; (3) quinine in benign and cases of moderate severity, and daily

cold baths, 68° to 86° F., in severe cases, 7.33 per cent. These to be considered in connection with Brand's method, 9.92 per cent. mortality. The authors admit that the method which is apparently gaining ground is that of Brand, due largely to the enthusiasm of its adepts, its apparent simplicity, its inflexible rules which relieve the practitioner of all investigation as to indication in obliging him to follow a route which is mathematically worked out. It is fair to recognize its success in severe and malignant cases, for which it should be exclusively reserved. With this chapter, a monograph in itself, we place the book upon our shelves conveniently for reference. The boundaries of medicine have so rapidly widened, the field has been so thoroughly tilled that no single brain can adequately present nor one book contain its well-proved facts. We trust that the succeeding volumes, with their numerous contributors, will maintain the high scientific standard, show the same conciseness, and aim to satisfy the necessities of the advanced physician as does this.

R. W. W.

DISEASES OF INFANCY AND CHILDHOOD. By L. EMMETT HOLT, M.D. 8vo., pp. 1117. New York: Appleton & Co., 1897.

ONE of the most striking of the many happy qualities of Dr. Holt's book is its completeness; another is its originality; and, perhaps equally prominent with either, is the care shown in its composition. The author has been a number of years in the actual construction of the volume, and has been peculiarly well fitted for his task by the most favorable opportunities for extended experience in the study of the diseases of children, both on the living subject and at the post-mortem table. If we add to these advantages a certain methodical quality of mind, with faculties of analysis and judgment and a knowledge of the needs of the student, we find all the chief requirements for the production of a successful book. That the work is an excellent one, even a superficial glance shows, and careful reading of it adds to one's good opinion. The presswork is good, the illustrations as numerous as the requirements demand, among them being a number of colored plates.

Looking more particularly into the subject-matter of the volume, we find it difficult to pick out any subject for special praise as compared with others. Perhaps most noteworthy is the section on feeding of infants, which is an elaborate study, with special emphasis upon the modifications of milk to produce definite percentages of the normal constituents. This section will be found invaluable by physicians wishing to practise home modification of milk in a thoroughly scientific manner.

Useful, too, and eminently practical, are the introductory remarks on the hygiene and care of children, on growth and development of the body, and on peculiarities of disease in children. The section on the diseases of the digestive system is very satisfactory. It contains, among other matters, a series of illustrations showing the size of the stomach at different ages, with useful remarks on the physiology of digestion in infancy. We note with especial pleasure a very satisfactory article upon cyclic vomiting, regarding which little or nothing is to be found in most text-books. The remarks on "acute gastro-enteric infection," as the

author designates summer diarrhœa and allied forms, are thoroughly up to date. The photographs and drawings in the article on colitis and ileocolitis are exceedingly fine. The article on stomatitis leaves one somewhat in doubt regarding the nosological position of some of the instances of house epidemics of an ulcerative disease of the mouth which are occasionally seen. We note also the statement, with which certainly all writers are not in accord, that quinsy is always a unilateral disease.

In the chapter on respiratory affections we observe with particular pleasure the beautiful photo-micrographs of pneumonia of different forms. The article upon this disease is very complete.

Diseases of the circulatory apparatus open with one of the clearest descriptions of congenital cardiac lesions and their symptoms with which we are acquainted.

To review at length the remaining sections of the volume would serve no good purpose. We may merely mention in passing the very useful general remarks on the character, quality, and method of collecting urine in infancy, and on some of the minor affections of the urinary apparatus, often neglected by writers; the articles on convulsions, meningitis, cerebral tumor, and adenitis; and the section on infectious diseases, particularly upon diphtheria. We fail to find any mention of the family form of spastic paraplegia, either of the cerebral or spinal type, and we regret to see anterior poliomyelitis called "infantile spinal paralysis," as this seems a step backward in exactness of nomenclature. Glandular fever is not spoken of—perhaps intentionally. The term "anginose" is omitted in writing of the form of scarlet fever usually described under this title. Erythema exudativa (on page 815) should, of course, read erythema exudativum.

Yet these criticisms are merely upon secondary matters. As to the work as a whole, it is a sincere pleasure to possess it, to read it, and to realize that it is such a credit to American medical book-making.

J. P. C. G.

MITTEILUNGEN AUS DEN GRENZEBIETEN DER MEDIZIN UND CHIRURGIE. Redigiert von J. MIKULICZ (Breslau) and B. NAUNYN (Strasburg). Erster Band. Drittes Heft. Jena: Verlag von Gustav Fischer, 1896.

CONTRIBUTIONS FROM THE BORDERLAND OF MEDICINE AND SURGERY. By J. MIKULICZ and B. NAUNYN

NOT long since (AMERICAN JOURNAL OF THE MEDICAL SCIENCES, April, 1897), we noticed one part of the first volume of this excellent work. Another part reaches us now with ten contributions instead of a single one. We can only give a few notes of some of the more important.

The first paper is by Bandler, on The Influence of Chloroform and Ether on the Liver. He reports the case of a man who was operated on for hernia, the operation lasting an hour, chloroform being used as the anæsthetic, and no iodide or bichloride being used. On the day after the operation he developed jaundice, and on the fourth day died soporose. The post-mortem examination disclosed the fact that he died with acute yellow atrophy of the liver, together with a fatty heart and chronic Bright's disease. Bacteriological infection was excluded at the post-mortem; no other cause apparently existed, therefore, except the chloroform.

Many years ago Casper pointed out the possibility of such after-results from chloroform, and the investigation made by the author on dogs, as well as the post-mortem on his patient, seemed to establish the fact that occasionally such a result may follow.

The next article is by von Jaksch, on The Treatment of Malignant Tumors with the Erysipelas Serum of Emmerich-Scholl. The conclusions which he reached are very much the same which both experience and observation have impressed upon our own mind as to the use of the erysipelas serum of Coley. The author says "the present serum does not enable us to reach our object. Its employment brings dangers and great suffering for the patients, so that after the large number of serious accidents following its use in a relatively small number of cases I cannot advise its further clinical use."

Siegel and Heinshaimer complete two interesting articles on operations on the stomach.

Gerhardt and Lauenstein have papers on Perityphlitis. It seems strange that eleven years after Fitz's convincing paper our brethren, both in England and especially, perhaps, those in Germany, still adhere to the pathologically erroneous name perityphlitis.

Franke reports an interesting case of Recovery from Mucous Colitis by the Establishment of an Artificial Anus. Our own experience as to the ease with which patients can take care of an artificial inguinal anus and the comfort which they derive from such operative procedures in suitable cases makes us very much more willing than formerly to use this method in the treatment of cases of such gravity as to demand it. Such patients can go about with ease and comfort not only to themselves, but to all about them, since no one would know that an artificial anus had been established in the majority of the cases.

In the paper of Berkhan on the Treatment of a Diverticulum of the Œsophagus, we cannot but be struck with the immense superiority of the surgical over the medical treatment. A clergyman, aged fifty-two years, was under treatment for three months. The œsophagus was dilated by a bougie of suitable curve, which, however, it took eight days of repeated experiment to pass into the stomach. After that the bougie was introduced eighty-five times, and the patient declared that since the beginning of the treatment he had never been able to swallow so well. He wished to learn the technique of the operation himself, so that he could resume his occupation without requiring the constant aid of the surgeon. A curious commentary on this statement follows. Before this suggestion could be carried into effect it became entirely impossible for him to swallow, and after several days of rectal feeding the patient died exhausted.

A case of Professor Hearn, on which he operated at the Jefferson Medical College Hospital, presented a very different and striking contrast. The diverticulum was exposed by a vertical incision at the side of the neck; it was cut away, the margins were inverted and united by Lembert sutures, precisely as in any other part of the digestive tract; primary union took place; in a week the patient was up and about, and has had no trouble since. Surely with such a contrast, especially with the death of the one patient after three months of treatment, and the entire recovery of the other in one week, the choice of methods is easily made.

Two other articles, on Wandering Neuritis after Traumatism, by Krehl, and a case of Hæmatomyelia Combined with Traumatic Spondylitis, by Henle, complete this interesting number. W. W. K.

A System of Practical Therapeutics. Edited by Hobart Amory Hare, M.D., Professor of Therapeutics and Materia Medica in the Jefferson Medical College of Philadelphia; Physician to the Jefferson Medical College Hospital. Vol. IV. With illustrations. Pp. ix., 1062. Philadelphia and New York: Lea Brothers & Co., 1897.

The purpose of this volume is to present the real advances which have been made in the treatment of disease since the three composing the system have been completed. It practically not only brings the work down to the present, but, on account of many changes among the sub-editors, gives a broader treatment of some of the subjects.

We are in accord with Dr. Simon Baruch in claiming great advantage in the use of public rain-baths, and welcome their more extensive introduction. The sentence "the *public rain-bath* has also been freely ventilated" is scarcely felicitous. As for the rest of "Hydrotherapy in Disease," we find the more recent contributions considered, but the very important paper of Porter omitted. We appreciate the arguments for the method, and feel that if its value should not receive fuller recognition it will not be for lack of energy or enthusiasm on the part of its devotees. Under the heading of "Mineral Springs," Bad Nauheim receives much attention, but is more fully discussed by Henry (pp. 379 *et seq.*). Dr. S. Edwin Solly reviews the "Present Treatment of Tuberculosis," taking up tuberculin, anti-streptococcal injections, nuclein, antiseptic, and other drug treatment. So far as concerns creosote, we believe the author to be in the wrong when he assumes that the good results obtained from this drug to be due to the fact that only patients with good digestion can take it, and others are dropped from the roll, and thus are not counted. There are creosote preparations which are readily taken by persons whose digestions are impaired, so that we believe that the fault is not with creosote, but with the preparation. The monograph of Audeoud is too valuable that he should appear as Audeon (page 48). We agree with the editor in preferring camphoric acid for night-sweating to zinc, recommended by the author; this in our experience may be effective, but most certainly interferes with digestion. The question of climate is distinctively within the province of the author, and we note with pleasure his very fair presentation and sound advice, to which even more space could have been advantageously given. Dr. Edward Martin offers a practical chapter upon the "Present Treatment of Syphilis," to which no exceptions should be taken, for its value is that of a monograph. "Typhoid Fever and Malarial Diseases" have been entrusted to Dr. J. M. Anders. He reviews hydrotherapy fairly, recognizing what many of its followers apparently ignore, "the supreme importance of adapting the means to be employed to the case in hand rather than to the disease in general." Of special medication the author speaks with indefiniteness, although offering some important suggestions. In the second division of the subject we find nothing of especial novelty.

The editor, Dr. H. A. Hare, presents the subjects of "Influenza, Scarlet Fever and Measles, Diabetes Mellitus, and Intestinal Parasites." We confess our surprise that the bacillus of Pfeiffer is not recognized as associated with influenza, especially since the work of Canon has been published, and that Ingals, who takes up this disease from another standpoint, apparently is of the same opinion. But this does not lessen our appreciation of his (Hare's) sound, practical treatment of this subject, nor of the next, which is scarlet fever. Of measles, however, we think more might have been said. If the author is correct in his statement that "the necessity for treatment of measles is usually in direct proportion to the bad handling of the case from a hygienic point of view, unless the attack be uncommonly severe or the patient be already weak and feeble, and so unable to resist disease," should we not justly expect more than a half page upon which to point out the hygienic precautions which, omitted, result in "bad handling?" We criticise this defect not because of the lack of information on the part of the author, but because he is not just to his reputation as to thoroughness. Dr. E. Fletcher Ingals presents an exhaustive chapter upon the "Diseases of the Nasal Chambers and Associated Affections," quite profusely illustrated. Dr. D. Braden Kyle follows with a fair discussion of the "Diseases of the Uvula, Pharynx, and Larynx." "New Facts and Methods in the Treatment of Diphtheria" is the subject assigned to Dr. William Halleck Park, which comes to us in fairness and conciseness. We would have more fully indorsed his conclusions had Health Board figures, so notoriously untrustworthy, been eliminated. As it now stands, the horse is apparently the source of the greatest danger in the antitoxin treatment. Dr. Norman Bridge takes up "Asthma, Bronchitis, and Whooping-cough" in a very interesting manner. "Pleural Effusion and Empyema, Abscess, and Gangrene of the Lung" is entrusted to Dr. A. J. McCosh. This should be read in connection with the following chapter, that a complete presentation may be obtained. This is by Dr. James B. Herrick, and is entitled "Pneumonia, Croupous and Catarrhal, and Pleurisy." The treatment of pneumonia is rather a *résumé* of modern methods than of recent progress, and we are of the opinion that a proper consideration should include not only problems from the bacteriological, but as well from the mechanical standpoint. We deem the chapter a suggestive one, and worthy of study and subsequent reflection. Dr. Frederick P. Henry takes up the "Diseases of the Heart" in a thoroughly systematic manner. He has not advanced the method of baths and resistive exercises as a panacea for all circulatory disturbances, apparently recognizing that some of the British monographs upon the subject unconsciously are contributing to the amusement of the thoughtful physician. Dr. Ralph Stockman contributes a short but satisfactory article upon the "Diseases of the Blood," while the "Diseases of the Liver" receive inadequate notice by Dr. Joseph Eichberg. Dr. S. J. Meltzer not only gives us a readable chapter upon "Diseases of the Thyroid and Thymus Glands, Myxœdema, Cretinism, Graves's Disease, and Obesity," but adds a very creditable bibliography. Dr. Thomas G. Ashton presents "Diseases of the Stomach" in a way which shows his thorough acquaintance with the literature, although the practical physician may wish for definiteness in some statements. Dr. George Ryerson Fowler goes over the subject of "Peritonitis" and its allied conditions, including "Obstruction of the Bowels,"

without presenting much of novelty. His advice seems to be sound from the physician's standpoint. Dr. Joseph M. Matthews gives an exhaustive study of the "Diseases of the Rectum and Anus." "Diarrhœal Diseases and Dysentery," by Dr. W. W. Johnston, is far too brief, discursive, and scarcely satisfactory. "The Modern Treatment of Diseases of the Skin," by Dr. H. W. Stelwagon, is rightly entitled, although we wonder how scorbutus came to be assigned to a dermatologist when the skin manifestations, if the most striking, are certainly not the most important. For clearly cut, positive statements, Dr. Joseph Collins on the "Spasmodic Affections of the Nervous System" should be commended. The statements are not all new, but the treatment of the subject is original, and every page bears the impress of the author's personality. A very much needed chapter is that upon "Drug Habits," by Dr. F. X. Dercum, who is brief but satisfactory. Dr. Hugh T. Patrick, on the "Disorders of Sleep," gives the reader valuable distinctions between different states and important therapeutic suggestions. Dr. Nathan S. Davis, Jr., takes up the subject of "Therapeutics of Renal Diseases." While there is much of importance, we believe that the time has come when renal degenerations should be sharply separated from inflammations. "The Therapeutics of the Diseases of the Female and Male Genito-urinary Tract" is by Drs. E. E. Montgomey and William T. Belfield, respectively, and both traverse the border-line of surgery. Dr. Casey A. Wood takes up the "Treatment of Diseases of the Eye," and Dr. S. MacCuen Smith "The Ear," from the standpoint of the general practitioner; the former is noticeable for the number of prescriptions.

We have noticed the different authors with more than usual completeness, for, unlike most conjoint works, this does not contain a hopelessly bad chapter. We congratulate the editor that he has been able to secure such varied and representative ability to the end that this volume represents the present status of therapeutics, and, further, that his supervision has resulted in so few instances of contradictions and overlapping. We predict for this volume a success equally satisfactory as that accorded to its predecessors, for we believe it is deserved. R. W. W.

PROGRESS

OF

MEDICAL SCIENCE.

THERAPEUTICS.

UNDER THE CHARGE OF

REYNOLD W. WILCOX, M.D., LL.D.,

PROFESSOR OF MEDICINE AND THERAPEUTICS AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL; VISITING PHYSICIAN TO ST. MARK'S HOSPITAL.

The Local Action of Tetanus Toxin.—DR. R. L. PITFIELD, having injected this substance into the calf of his left leg, found that it was more irritating than an ordinary hypodermatic injection, and the parts became rapidly more and more painful. An hour afterward the leg hurt considerably; there was no increase in any reflexes; the body-temperature had risen one degree above normal; there were headache and giddiness. Two hours later the temperature fell to normal, but the pain became intense; there was spasm of the fibres of the muscles, which became greatly aggravated with use. The only manifestation of the poison was local, but it was most severe, and lasted twelve hours. It is suggested that this treatment might be of use in subacute or chronic myelitis, especially in children. The patients, of course, would gradually become immune to the poison. In case of accident the antitoxin could be used.—*Therapeutic Gazette*, 1897, No. 3 p. 145.

Antistreptococcic Serum in Severe Puerperal Septicæmia.—DR. ARTHUR W. SHARP reports a single successful case. In this there were injections of two and one-half drachms on the fifth, sixth, and seventh days, and of an ounce on the eighth day of the disease. Convalescence was established upon the fifty-third day, although the temperature reached the normal at the thirtieth. The serum was injected into the abdominal fat and the following precautions taken: syringe and needles boiled, skin scrubbed with soft soap, then with 2 per cent. solution of lysol; next dusted with boric acid, and finally a dressing was applied. No inconvenience whatever resulted from any of the punctures, and the patient's appetite was markedly stimulated. The following conclusions are presented: (1) If proper precautions are taken, the risks attending the use of the serum may be neglected. (2) Although no streptococci were found in the blood, benefit attended the use of the serum. (3) In really serious septic cases corrosive sublimate (1 to 2000),

used fearlessly, is the only reliable antiseptic. (4) In septicæmia after labor the focus of mischief may be other than uterine.—*British Medical Journal*, 1897, No. 187, p. 519.

Traumatic Tetanus.—Dr. Ludwig Rabek reports a single successful instance of the use of antitetanin. The immunizing strength was one thousand millionth of the body-weight. The dose of this was from 12 to 24 drachms. One week after the injury characteristic symptoms supervened, and the treatment was begun a week later with the minimum dose. On the following day the maximum dose was given and repeated four days later. Convalescence was established at the end of a week, although the abscess resulting from the second injection required two weeks more for its healing. The age of the boy (six years) and the rapid improvement add to the interest of the case.—*Therapeutische Wochenschrift*, 1897, No. 10, S. 226.

[The value of the remedy is not so clearly demonstrated in chronic as in acute cases, yet the reported successes belong chiefly to the latter class.—R. W. W.]

Holocaine, a New Anæsthetic.—Dr. G. Gutmann presents this, which is p-diæthoxyæthenydiphenylamidin, as an advantageous substitute for cocaine. This is a crystalline base from which is formed a chloride, the latter soluble up to 2½ per cent. in cold water. Boiling does not decompose a 1 per cent. solution, which is also permanent even if left in open vessels. This is the solution employed in thirty cases. Two to five drops are instilled, causing a slight burning sensation which disappears within a minute. Three to five drops produce within a minute a total anæsthesia of the cornea which lasts, upon an average, nine minutes, during which the corneal surface remains moist and clear. In one instance four drops produced so complete an anæsthesia that the galvanocautery was used, without pain, upon a corneal ulcer. In all cases the tension was unchanged, and the pupils showed neither myosis nor mydriasis, nor was the accommodation altered. The disadvantages of this anæsthetic are its limitation to operations where its effect can be obtained by instillation, for when administered hypodermatically it is poisonous. Further, it can be sterilized only in vessels which are free from alkali; those of porcelain are recommended.—*Deutsche medicinische Wochenschrift*, 1897, No. 11, S. 165.

Antipneumococcic Serum.—Dr. J. W. Washbourn gives an account of the immunization of a pony, and reports two cases of pneumonia treated with the serum. The pony was first injected, into the subcutaneous tissue of the shoulder, with broth cultivations of the pneumococcus heated to 140° F. for one hour, then with living agar cultivations, and lastly with living broth cultivations. The injections lasted for four months. Five months after the last injection the animal was bled and the serum found to possess marked protective properties. Having obtained a serum of known strength, he made use of this in two cases. Of these he remarks that the injection of the serum in the first was followed on both occasions by a fall of temperature, and on the day after the second injection the crisis occurred. Inasmuch as this was the ninth day of the disease, it is difficult to say

whether the result was due to the treatment or not. The attack was a fairly severe one, but the pulse was good before the serum was administered. The second patient, though young, was a bad subject for pneumonia on account of his intemperate habits. The attack was a severe one, and both lungs were affected. There was a good deal of delirium; the pulse was 140, and the heart showed signs of dilatation on the second day of the disease. The serum appears to have exerted a beneficial effect upon the disease. On the day after the first injection the pulse-rate had fallen from 132 to 112. The process in the left lower lobe never advanced after the treatment was begun. It will be observed that the serum was injected at an early stage—namely, on the third day of the disease. In both cases an urticarial rash was observed during convalescence, similar to the rashes occurring after injection of antitoxic and antistreptococcic sera. Since no satisfactory toxin has been obtained from cultivations of the pneumococcus, it is impossible to say whether this serum possesses any antitoxic power. The serum should be injected into the subcutaneous tissue, and strict aseptic precautions taken. The skin should be well washed with an antiseptic and the syringe well boiled immediately before use. Five drachms (660 units) should be injected, twice daily, until the patient is convalescent, commencing as early as is possible.—*British Medical Journal*, 1897, No. 1887, p. 510.

[Those who consult the original should note that the first temperature-chart is reversed.—R. W. W.]

Parthenium Hysterophorus.—Dr. H. V. Arny gives a careful botanical description and chemical analysis of this plant, which is found in great abundance in Jamaica and Louisiana. Although no alkaloid or glucoside was found by the author, Ulrici claims an alkaloid which he named parthenicine. There was isolated, however, a substance, which is named parthenin, a proximate principle, varying in amount according to time of collection, but reaching the maximum in July (1.13 per cent.). Tovar reported it as a remedy for facial neuralgia, and it also proved beneficial in a case of fever and anæmia where quinine failed. Guyer confirmed its efficiency in neuralgia, especially of the cranial variety, but found it utterly without effect as an antipyretic. In Jamaica the plant is used as a remedy in ulcerated sores and in herpetic or pustular skin diseases. Of parthenicine, grain doses may be given every hour in neuralgia, while fifteen grains are used in intermittent fever.—*American Journal of Pharmacy*, 1897, No. 4, p. 169.

Spermin.—Dr. G. Hirsch quotes the statement of Poehl (1892) that, without exception, all organs used in therapeutics contain spermin, which plays the part of a physiological-chemical ferment of tissue-respiration and outside the body sets in motion oxidation-processes. Instances of its use in anæmia, tabes, and endarteritis obliterans are reported, and various authors cited, from which the conclusion is reached that the drug has no specific action in a given disease, but rather that it regulates internal oxidation, increases metabolism, removes excrementitious products which injure the nervous system, and gives the *vis medicatrix naturæ* undisturbed operation.—*St. Petersburger medicinische Wochenschrift*, 1897, No. 7, S. 51.

Gelsemium.—Prof. L. E. Sayre has made a careful microscopical study of the root, and has come to the conclusion that the drug as found in the market is composed not only of the rhizome and root, but also of the stem, in varying proportions; the stem is an adulterant, since it has no medicinal value. An analysis of these parts is promised by the author, and when completed we are likely to know the reason for the great variations in therapeutical activity so often observed.—*American Journal of Pharmacy*, 1897, No. 1, p. 8.

Camphoric Acid in the Treatment of Night-sweats.—Dr. H. A. Hare finds that this remedy controls the sweating of tuberculous patients in the great majority of cases and does not produce any disagreeable symptoms, such as are usually caused by atropine or other powerful antisudorifics. Twenty grains are ordinarily quite sufficient to control the sweat, provided the dose is given early enough to be absorbed before the time at which the sweating appears. Other observers have shown that as much as sixty grains may be given without any deleterious effect. The drug may be given in cachet, dissolved in whiskey or brandy, placed in dry powder upon the tongue and washed down with a little water or milk; but as it is slowly absorbed, it should be given an hour or two before the anticipated time of the sweating.—*Therapeutic Gazette*, 1897, No. 3, p. 164.

Treatment of Leprosy.—Dr. Domenico Fornara practises inunction and injections of airol [bismuth oxy-iodogallate]. The inunctions are of 10 per cent. solutions of the drug in vaseline, which give also the beneficial results from massage. For injections into the leproma the following formula is employed: aristol, 5; glycerin, 35; distilled water, 10. The inunction is performed over the whole body at night; the next morning the patient takes a warm bath, and during the day the *plaques* and tubercles are injected. For the nose and throat the powder is insufflated, or, in case this is not well borne, the parts are painted with the ointment. The drug is undoubtedly absorbed and enters the blood as iodine, bismuth, and gallic acid, in a nascent state, and this gives rise to a vigorous antiseptic action. During the early days of its administration by subcutaneous injection there appear dyspnœa, feeble heart-action, and reversed gastric peristalsis. Later appears a gray line at the junction of the gums with the teeth, which is attributable to the bismuth, while the other symptoms are caused by the gallic acid. —*Therapeutische Wochenschrift*, 1897, No. 12, S. 71.

Treatment of Chlorosis.—Dr. H. Huchard divides the patients into three classes, with whom (1) iron is useless, (2) harmful, or (3) very useful. Chlorotics of the first degree (Hayem) need rest, proper diet, and country air, and iron is useless. In the second class should be placed the dyspeptic chlorotics who may suffer from *hyper-* or *hypochlorhydrie*, gastric dilatation, gastro-intestinal fermentations, or gastric ulcer; here the dyspepsia must be treated first; but if iron is first administered, it will be found that it is harmful. Iron will be useful in these patients later, when administered as an oxalate or lactate up to five or seven grains daily. For the irritable chlorotics both sea-air and high altitudes are objectionable, as are also long

journeys. For this class Gérard-mer, La Bourboule, Evian, or Saint-Gervais is indicated. The torpid chlorotics require sea-air or the muriated arsenical waters of Bourboule or weak sulphur waters. The treatment is summed up in three words—rest, air, and iron. Much of the first two, but little of the last.—*Bulletin Général de Thérapeutique*, 1897, 5e liv. p. 149.

The Use of Iodothyrin in Diseases of Children.—Dr. Friedrich Lanz reports the results of the use of this remedy in twelve cases of struma. From these he concludes that this is a remedy for parenchymatous struma; in comparison with other iodine preparations this contains as much as the fresh thyroid gland, and relieves in a much shorter time, and only exceptionally presents disadvantages, and these of slight importance. With this, as with iodine, its disuse results in relapse.—*Therapeutische Wochenschrift*, 1897, No. 11, S. 247.

The Use of Levulose in Diabetes.—Drs. E. de Renzi and E. Reale found that eight diabetic patients could completely oxidize this substance in daily amounts of from six to twenty-five drachms. In artificial diabetes (dogs after extirpation of the pancreas) no sugar of any sort was found in the urine after its administration. Further than this the use of levulose not only does not increase, but rather diminishes the amount of nitrogenous output, both urine and feces being examined—*Wiener medizinische Wochenschrift*, 1897, No. 9, S. 379.

Diabetes.—Dr. T. H. Kellogg states that the simple form is readily controlled by restriction of the dietary and general increase of oxidation-processes by means of vigorous Swedish movements, cool shower-baths followed by brisk rubbing, the Scotch douche, and the inhalation of oxygen. If there is an excessive amount of organic waste, as shown by increased nitrogen oxidation, the patient must rest in bed and avoid massage and passive movements. Warm baths, tepid sponging, and tonic applications of electricity should be administered. The diet should consist chiefly of buttermilk and well-cooked preparations of milk. An excessive meat-diet is objectionable in all forms, especially in the grave. If there is diminished oxidation of proteids, there is special danger that the system will not relieve itself of the normal tissue-poisons, so that the addition of toxic elements, which are always present in the flesh of dead animals, may precipitate an attack of coma. Restriction to a diet absolutely free from starch is not required, as the system requires a certain amount of carbohydrates. The gluten of wheat, either pure or with the greater portion of the starch removed, is an invaluable food. Most of the gluten flour and preparations are fraudulent in that they contain practically no less starch than is found in ordinary wheat-flour. It is believed that soja beans, which contain only 11 per cent. of starch and an equal amount of sugar, with 35 per cent. of proteid matter in the form of true albuminoids, and 1 per cent. of a very digestible fat, will be found useful in this condition.—*Modern Medicine*, 1897, No. 3, p. 57.

The Physiological Action of the Cold-bath Treatment—Dr. William Henry Porter credits Mathewson as among the first (1860) in this country

to use the application of cold to the surface of the body with a view to lowering temperature. When the fever-heated patient is stripped and wrapped in a cold sheet, or sponged with tepid or cold water, alcohol and water, or is placed in a cold bath, these cause a decided increase in the radiation or excretion of heat from the surface of the body, and cool the blood circulating in the cutaneous capillary bloodvessels; thus the blood sent back to the deep or excretory organs is a shade cooler than obtains before the bath. These changes do not increase the excretion of the toxins; therefore the result of the bath, if this is all that can be accomplished by its use, is to subject the individual to great discomfort, and lower the surface-temperature for a very short time, without materially benefiting the pathological condition. The stimulating action of cold applied to the skin in this manner causes a contraction of the surface bloodvessels, and a large volume of blood is thereby transferred from the capillaries of the surface of the body to those that go to make up the splanchnic arcade and those of the deep and excreting organs. Thus the liver and kidneys are at once the seat of a large afflux of blood from the surface. When this change is brought about quickly, as it usually is, and the force of the heart is insufficient to fully sustain the sudden shifting of so large a bulk of blood from one vascular arcade to another, there result an intense congestion of the liver and kidneys, a slowing or arrest of the circulation in the kidneys, and a suspension of their functions, a great accumulation of toxins within the body, intense poisoning of the nervous system, followed in short order by death, which in this instance is directly traceable to the cold bath. If, on the other hand, the heart is sufficiently strong and properly sustained before, during, and after the bath, and provided the patient is also vigorously rubbed or massaged while in the bath, the congestion of the liver and kidneys may be quite as intense, but it is not followed by stagnation of blood in these organs and arrest of their functions. The circulation is actively maintained and the special toxins of disease and the faulty products of metabolism are more rapidly eliminated. The application of cold to the surface of the body, together with friction, acts reflexly upon the deep excretory organs, thus enabling the glandular system to maintain a better nutritive condition. The cold bath should be employed early in the disease, before the glandular organs have become badly damaged and prior to the intense weakening of the heart and the exhaustion of the nervous system. He denied that he was a strong advocate of the cold-bath treatment, for he had not placed a typhoid fever patient in a cold bath since 1887. This patient died of renal congestion and uræmia, probably hastened by the cold bath. Since so much has been recently said upon bath treatment, it seemed that inquiry into its physiological action would be interesting.—*The Postgraduate*, 1897, No. 3, pp. 113 and 156.

Poisoning by Potassium Chlorate.—Dr. Rechstomer notes the instance reported by Romanow, of a woman of twenty-five years, who absorbed three ounces of this drug with suicidal intent. Death followed in six days. The necropsy disclosed fatty emboli in all the organs, especially in the small vessels and capillaries of the lungs. The author quoted, believing that under the influence of this poison the marrow of the long bones would be pro-

foundly changed, undertook some experiments upon dogs. These animals presented the same fatty emboli, but less generalized. Particularly was it noted that they were found in the capillaries and small arteries, and also large cells similar to those of the bone-marrow —*Journal des Praticiens*, 1897, No. 1, p. 14.

The Treatment of Syphilis by Hypodermatic Injections.—Dr. Josef Grünfeld, so far as concerns ambulant patients, believes that this method is preferable to all others in the treatment of constitutional syphilis. The deep intermuscular injections of large single doses under antiseptic precautions is not followed by unpleasant complications or results. The position in this method is satisfactory, yet it does not secure the rapid curative effects of inunction. The formula which seems to be preferred is: corrosive mercuric chloride, 1.25; sodium chloride, 3; distilled water, 50.—*Centralblatt für die Gesammte Therapie*, 1896, Heft xii. S. 705.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

PROFESSOR OF MEDICINE IN THE JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND;

AND

GEORGE DOCK, M.D.,

PROFESSOR OF MEDICINE IN THE UNIVERSITY OF MICHIGAN.

Malarial Hæmoglobinuria.—Bastianelli (*Le Emoglobinuria da Malaria, secondo i recenti Studi; Annali di Medicina*, Anno ii. Fasc. xi.) has recently contributed an excellent article upon malarial hæmoglobinuria, based not only upon the cases observed in Rome, but upon the careful analysis of the work of others. He asserts that it is practially proved that hæmoglobinuria occurs only in infections with the æstivo-autumnal parasite. The recent observations of A. Plehn tend to show that the cases analyzed by F. Plehn, as well as his own, were instances of æstivo-autumnal infection.

The manifestation is rare in Italy, commoner in Sicily and Greece, very common in the tropics. An interesting point is that hæmoglobinuria following quinine is extremely rare in Italy, no case having ever been reported from the Campagna. The frequency with which these cases occur increases, just as in cases of spontaneous hæmoglobinuria, as one passes South.

Hæmoglobinuria due to quinine never occurs excepting in patients who are suffering or who have recently suffered from malarial fever.

Spontaneous hæmoglobinuria may be at times intermittent, or, as is more common, in single paroxysms. He divides the spontaneous hæmoglobinurias into three classes:

(*a*) In the blood there are found æstivo-autumnal parasites of the pyrogenic

cycle—that is, young hyaline forms. Here the hæmoglobinuria occurs regularly in association with the segmentation of a group of parasites—that is, at the time of the ordinary paroxysm. This he terms the paroxysmal hæmoglobinuria.

(*b*) In the blood there are to be found only crescentic or ovoid bodies and pigmented leucocytes, or, possibly, pigmented leucocytes alone. Some of these patients come to autopsy while the blood is quite negative as regards parasites. They may also be wanting in the internal organs, but from the intravascular melanosis it may be seen that the parasites have disappeared quite recently. In this class the paroxysm of hæmoglobinuria is caused by the segmentation of a group of parasites which disappear spontaneously during the attack. The changes left in the blood and internal organs, however, are so great that the paroxysm continues for hours or days after the organisms have disappeared from the blood. Bastianelli terms this form the post-paroxysmal hæmoglobinuria.

(*c*) The blood examination is entirely negative, and the only evidence of there having been a malarial infection is the presence of an endothelial, perilobular, perivascular melanosis. The malarial process has come to an end some time previously. Here the attack of spontaneous hæmoglobinuria does not depend upon the presence of parasites, but starts without apparent cause. Such paroxysms may be intermittent and repeated at short intervals, but are more commonly single, of long duration and very severe. This form is especially grave, and is the "pernicious hæmoglobinuria" of Marchiafava, while Bastianelli calls it the post-malarial hæmoglobinuria.

As to the causation of the hæmoglobinuria, in all probability the parasites which produce the febrile paroxysm play an important part in some cases. An anæmic condition of the blood, however, seems to be indispensable for the production of the phenomenon, as the hæmoglobinuria does not occur with the first paroxysm, either in the initial attack or in a relapse. It is rare under any circumstances in the first attack, usually occurring with the first relapse or after repeated relapses. There is also in some cases an individual idiosyncrasy or predisposition.

Hæmoglobinuria does not occur in the most chronic cases of malaria—that is, in those cases where a certain equilibrium is established between the needs of the organism and the function of the hæmatopoietic organs—but in those cases where the organism is still liberating itself from the residue of the infection and in which anæmia and melanosis still exist.

Bastianelli, finally, takes up the hæmoglobinuria due to quinine. He asserts that:

(1) It occurs only in individuals in whom a malarial infection has preceded.

(2) The hæmoglobinuric attack is produced constantly every time that quinine is administered, whether it be given while the malarial attack is in progress (Tomaselli), or whether it be given when the malarial infection has run its course (Murri).

(3) Extremely small doses are capable of bringing on an attack.

(5) Quinine hæmoglobinuria has been seen in patients who have already suffered from spontaneous hæmoglobinuria (Murri).

Quinine hæmoglobinuria is rare, excepting in the tropics. No doubt there exist cases of hæmoglobinuria due distinctly to the administration of quinine.

Bastianelli says: "The preceding malaria creates the fundamental disposition; the existing malaria the accidental disposition; the quinine the provocative agent."

He divides quinine hæmoglobinuria into two forms:

(1) That occurring during the paroxysm—paroxysmal quinine hæmoglobinuria.

(2) Post-malarial quinine hæmoglobinuria.

In these varieties quinine, through a very considerable length of time, will produce a hæmoglobinuria whenever administered. There are, however, instances where the hæmoglobinuria due to the taking of quinine occurs only now and then during the paroxysm—that is, is an episode. These cases are rare.

Bastianelli comes to interesting and practical results with regard to treatment. The course pursued depends upon the blood examination.

If hæmoglobinuria occurs during a malarial paroxysm and parasites are found in the blood, quinine should always be given.

If, however, no parasites are found, either as a result of previous administration of quinine or on account of the spontaneous disappearance of the organisms, we may remember that the administration of quinine will have no effect upon this attack and that for the time being certainly another attack is not to be expected. In these cases Bastianelli recommends not giving quinine, owing to the possibility that the paroxysm may have been due to its previous administration.

If in an attack occurring in the middle of an ordinary malarial paroxysm there arises doubt as to its origin from quinine, it is well to abstain from the further administration of the remedy, for the quinine already given is usually sufficient to hinder the development of new febrile paroxysms. But if in a hæmoglobinuric attack which has come on after the giving of quinine the parasites are still found in the blood, one is justified, despite the danger, in insisting upon the specific treatment; if there be doubt as to the origin from quinine, we may be sure what the result will be if we allow the parasites to go on developing, and it is therefore safer to interfere.

[This careful abstract (for which we are indebted to Dr. Thayer) should prove of great value to those Southern practitioners who are interested in quinine hæmoglobinuria. The condition, so far as I know, is not met with in this latitude.—W. O.]

The Direction of Rotation in Cerebellar Affections.—Russell (*British Medical Journal*, April 10, 1897), in a previous communication to the *British Medical Journal*, May 18, 1895, published the results of experimental work on animals, in which he had found that animals deprived of one lateral lobe of the cerebellum rotated away from the side of the lesion. Dr. Bruce Ferguson believed that Russell had fallen into an error of expression, and concluded from the latter's description that the rotation was toward and not away from the side of the lesion. Russell, in the present article, endeavors to substantiate his previous views, and states that the discrepancy in views arises from the fact that some describe the direction in terms relating to the observer, while others describe it in terms relating to the observed. He believes that in the case of the human subject, as well as in animals, there

would be less likelihood of a difference of opinion if all physicians observed two rules: (1) That the object should be placed in front of the observer, and that both should be looking in the same direction. (2) That the direction of rotation about a horizontal axis should always be expressed in terms relating to the direction in which that part of the circumference of the object corresponding to its upper quadrant is moving.

Of all the methods of describing rotation, Russell thinks that the simplest is that in which the direction is compared to the rotation observed in a right-handed male screw. This being clear, the direction of rotation either resembles a screw entering or coming out of an object. The next in order of simplicity seems to be the method by which the direction is denoted by the terms "clockwise" and "anti-clockwise." It is necessary, however, in this method to note the relation of the object to the face of the clock.

Pathology and Etiology of Acute Ascending Spinal Paralysis.—KREWER (*Zeitschrift f. klin. Med.*, 1897, Band xxxii.) says that while most writers coincide in the view that Landry's paralysis is a multiple neuritis, every opportunity should be taken to study the changes in such cases. The writer reports four cases. In three of them, ending fatally, the peripheral and central nervous systems were studied microscopically. Changes were found in both. In the former a subacute degenerative neuritis; in the white substance of the cord a similar but more acute change; cloudy swelling of the cells in the gray matter, desquamation of the cells of the central canal. The vessels were distended with blood. No special changes in the cerebrum. No bacteria could be found in the sections. The author thinks that he can conclude that Landry's paralysis is nothing but the second and third stages of a chronic multiple neuritis that affects the cord by continuity. Here it spreads rapidly, generally upward, sometimes downward, causing death by involvement of the vital centres of the medulla. The outbreak usually follows an attack of some infectious disease. Clinically the progressive character of the paralysis is characteristic; the direction of its spread, however, is not. Pathologically, Landry's paralysis is a subacute chronic polyneuritis and an acute diffuse degenerative myelitis.

The Demonstration of Peptone (Albumoses) in the Urine and the Preparation of Urobilin.—SALKOWSKI (*Berl. klin. Wochenschr.*, 1897, No. 17) was struck by the fact that urobilin containing urine gave a violet color upon adding the first drops of copper sulphate of the Trommer test, and made a number of experiments to find if urobilin could give the albumose reactions. The material obtained by precipitating such urine with lead acetate and phosphomolybdic acid gave a biuret reaction and the urobilin absorption-band. The same is true of the residue obtained by shaking out in ether, making it probable that the biuret in the original comes from the urobilin. The desirability of having a preparation of urobilin for experimental purposes is evident. The method of Jaffe, modified by the use of phosphomolybdic acid, yields, the author thinks, a purer product than has yet been obtained. It differs from that obtained by the original Jaffe method in some of its physical characters, and its insolubility in ether. It gave a biuret reaction almost exactly like that of albumose.

Concerning the quantitative relations of urobilin, a 0.007 per cent. solution gives a very black absorption-band, and even a 0.003 per cent. solution a tolerably marked one. On the addition of zinc chloride to this latter solution (made alkaline) it becomes intensely rose-red, with marked green fluorescence. In a solution of this same strength made up with urine the urobilin can be shown both with the spectroscope and with zinc chloride. The stools may give an albumose reaction that is due to the content of urobilin.

The question as to whether other urinary pigments give such albumose reactions can be answered in the negative. Many urines with large urobilin content do not give the biuret reaction, probably because of the covering of the color by other pigments. Error is likely to arise only where the urine contains urobilin in quantities large enough to give a marked band in the spectrum. To detect albumose in the presence of urobilin it would seem that one had only to precipitate the urobilin with lead acetate and to test the filtrate for albumose. Unfortunately, the albumose may be carried down in the precipitate, and, if present in small amount, may escape detection. The same objection holds for such methods as decolorizing with charcoal and shaking out the acidulated urine with amyl alcohol.

As a rule, the author thinks it is best to precipitate at once with phosphomolybdic acid, because it is simpler, and small quantities of albumose are less likely to be overlooked.

A Case of Autochthonous Amœbic Dysentery.—Josef Sargo (*Wiener klin. Wochenschr.*, 1897, No. 18). There is no unanimity of opinion concerning the *rôle* of amœbæ in dysentery. Although it is not likely that dysentery will ever be proved to be a single disease, still much is to be hoped for from the new culture-methods. The various ways to account for the different forms by assuming different species of amœbæ or mixed amœbic and bacterial infections have none of them been established. The present case is of interest because of the rarity of sporadic dysentery in this latitude (Vienna) and on account of the results of inoculation.

Patient, male, aged twenty-seven years; no clear history of drinking foul water. The disease, which began with diarrhœa, followed by tenesmus and finally bloody stools, had lasted nine months when the patient first came under observation. Patient had lost weight, but had no disease in his other organs. Stools were frequent, mixed blood and mucus; they contained no pus. Amœbæ, containing vacuoles, red blood-corpuscles, and bacteria were found in large numbers. The patient recovered entirely in two months under irrigations of tannin and quinine.

The fresh stools injected into a cat caused bloody, amœba-containing stools, with temporary loss of weight. After the amœbæ disappeared from the stools, which, however, remained bloody, a second cat was inoculated from the first. This animal promptly got a bloody diarrhœa, but without loss of weight. The autopsies, four months and one and one-half months after the inoculations, showed the chief change to be in the lower part of the small intestine, where there was an enteritis, with involvement of the lymph-apparatus and swelling of the glands. There were ulcers with steep walls and much bloody, bile-stained mucus. No amœbæ. The part played by the amœbæ in the diarrhœas of the animals is not clear. The diarrhœa of the

second cat could not have been amœbic. These experiments do not point strongly to an amœbic cause for the dysentery of the patient. The ascending character of the disease in the small intestine may explain the intractable nature of the affection in man. In conclusion, the author suggests that heretofore a mistake has been made in killing experimental animals too early.

Chronic Diphtheria.—It is well known that diphtheria-bacilli may exist in the throat for months after an attack, and that they may occur in the healthy pharynx. Cases of chronic exudate are, however, much less common, and F. JENSEN (*Centralbl. f. innere Med.*, 1897, No. 19) reports the following: a nineteen-year-old servant-girl became ill with general symptoms and an ulcer on the right half of the soft palate, in the secretion of which virulent Loeffler's bacilli were found. During the next five months there continued to be an exudate in the pharynx in which virulent diphtheria-bacilli could always be demonstrated. The bacilli were characteristically influenced by the Behring serum, while it had no effect on the exudate. The best local results were given by a simple gargle of salt water. The blood-serum of the patient protected twenty times more than normal serum against injections of Loeffler's bacilli. The absence of symptoms of general intoxication during the greater part of the time was perhaps explained by the infiltration of the mucous membrane of the pharynx preventing absorption.

The Excretion of Uric Acid in Acute Pneumonia.—DUNIN and NOWACZEK (*Zeitschr. f. klin. Med.*, 1897, Bd. xxxii.). The authors review the theories that have been held concerning the origin of uric acid, and discuss at some length the process by which Horbaczewski arrives at his view that the uric acid is derived from the leucocytes and that it varies in amount with the number of leucocytes. This theory should allow of clinical demonstration. The authors began their work with the idea of proving clinically the correctness of Horbaczewski's claim. Pneumonia was chosen as affording the best opportunity, on account of the great hyperleucocytosis and the rapid absorption of the leucocyte containing exudate. Experiments were made upon five cases. In each the uric acid was estimated every day by the method of Haycroft, which has been shown to give results corresponding very nearly with those of the Salkowski method. The results show plainly that the uric acid is markedly increased during the absorption of the exudate. The uric acid began to increase the day before the crisis; the amount rose for two to four days, then fell slowly to normal in from three to four days more—a striking corroboration of the view of Horbaczewski.

Absorption from the Bladder.—MORRO and GAEBELEIN (*Zeitschr. f. klin. Med.*, Bd. xxxii.). Notwithstanding the work done on this subject, the results are contradictory, mostly due to faulty methods. Generally the experiments go to show that solutions of KI are absorbed. By inference this is true of other drugs. As regards the absorption of the normal constituents of the urine conclusions vary still more, Kaul claiming that they are and Treskin that they are not absorbed. The author's experiments were made upon dogs; the abdomen was opened and the ureters tied. The bladder was washed out and the carefully controlled solution injected. Later

the animal was killed and the fluid in the bladder again examined. The authors experimented with ten drugs, including cocaine, carbolic acid, and morphine; all except the last were absorbed. "The practical significance of the experiments is at once apparent, especially in the case of cocaine and carbolic acid. They show that in the local treatment of diseases of the bladder cocaine is only to be used with caution and carbolic acid not at all if severe poisoning is to be avoided."

SURGERY.

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

PROFESSOR OF CLINICAL SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE UNIVERSITY AND PHILADELPHIA HOSPITALS;

ASSISTED BY

ALFRED C. WOOD, M.D., INSTRUCTOR IN CLINICAL SURGERY, UNIVERSITY OF PENNSYLVANIA; ASSISTANT SURGEON, UNIVERSITY HOSPITAL.

AND

C. L. LEONARD, M.D., ASSISTANT INSTRUCTOR IN CLINICAL SURGERY IN THE UNIVERSITY OF PENNSYLVANIA.

A New Radical Operation for Inguinal Hernia.—Basing his departure on fifty-two operations which he has performed by Bassini's method, JONNESCO (*Cent. für Chir.*, March 27, 1897) modifies that operation in the following manner, with the purpose of obliterating not only the inguinal canal, but also the internal abdominal ring, by strengthening the abdominal wall at that point. He has performed this operation with perfect success in three cases:

The incision is the same as in Bassini's operation, commencing two finger-breadths from the pubic spine. The finger is then passed into the canal and its anterior wall divided. The hernia is reduced and the sac separated from the cord; it is much easier to isolate the hernial sac if it is first opened. The sac is ligated as high up as possible and the stump returned into the abdomen after resection

Before the cord is replaced beneath the peritoneum it should be carefully isolated, and care should be taken to see that the entire posterior wall of the inguinal canal is divided down to the peritoneum from the internal to the external ring. The cord is now replaced upon the peritoneum and a series of interrupted sutures introduced with a curved Reverdin needle. The first tissues united are, on the one side, Poupart's ligament, and, on the other, the internal oblique and transversalis aponeuroses. These sutures extend the entire length of the canal, closing the internal abdominal ring and extending to the pubic spine, where a sufficient opening is left to allow the exit of the cord, usually an opening that will admit the tip of the little finger. The second plane of sutures unites the muscular fibres of the internal oblique and transversalis to Poupart's ligament. These stitches take in the lower

tissues as well. The third plane of sutures unite the external oblique aponeurosis to Poupart's ligament, the stitches being placed deeply into the other layers. Toward the median line, where there are no fibres of the internal oblique, there are only two layers; here the aponeurosis of the external oblique is united to Poupart's ligament, the stitches including the posterior layer. At the external ring the two pillars of the external oblique are united, the stitches including the posterior layer and an opening being left for the exit of the cord.

The entire canal is thus closed and made firm by contracting scar-tissue.

The skin suture is in two layers, the first taking in the connective-tissue layer, the second approximating the skin edges, the object being to exclude any area where suppuration could take place. An antiseptic compressing-dressing is used to prevent the formation of a hæmatoma of the scrotum.

Sixty-one Cases of Partial and Complete Excision of the Tongue.—WHEELER (*Dublin Journal of Medical Sciences*, April, 1897) says that out of sixty-one operations performed by him for malignant disease, none died from the effects of the operation, save one, a male on whom he operated by Kocher's method modified; he died of asthenia. He did not tracheotomize him.

Of the sixty-one cases, in twenty-nine the disease returned. Many of these operations were performed not with a view to cure, but merely to relieve and remove a foul, sloughing, fetid mass, and thus render existence more tolerable. Twenty-two cases he was not able to trace at all directly himself. He heard indirectly that five were healthy three or four years after operation.

Seven others had had no return of the disease, to his own personal knowledge, one to fifteen years after operation, while three had no return seven, three, and two months after operation.

There is only one prognosis for unoperated lingual carcinoma—death; and a like prognosis for a large number of operated cases. That complete recovery does take place, he believes to be undoubted, as shown by his own cases.

The kindly healing of wounds in the mouth is not due to the bactericidal action of the saliva, but to the outpouring of leucocytes which its presence in these wounds always induces.

He considers it expedient to ligate the external carotid before removing the tongue, tonsils, and part of the palate; but preliminary ligature of the lingual arteries in excision of the tongue, as recommended by Mirault, is not required; there is no hemorrhage from the ranine arteries to cause alarm, and, as the operation upon the tongue is frequently postponed until the wounds for deligating the lingual vessels heal, the disease in the mouth is increasing and may be infecting the patient. The author believes it is good surgery to remove the tongue first and then the diseased glands later, if it is necessary to do the operation in two stages.

The inflammation in the glands usually subsides a little, and the glands can then be more readily removed, and the connective tissue will be a guide to the lymphatic glands deeper in the neck, where the lymphatics directly communicate.

The author has performed all the recent operations, but prefers the thermo-

cautery scissors when suitable. It is an admirable instrument in the majority of cases of lingual cancer where the disease is not too extensive. He never uses a ligature through the tongue. The tongue is held by toothed forceps, and just as the ranine arteries are about to be severed the hot blade is allowed to cool a little, in order to allow the arteries to bleed; they are then secured and ligated. Frequent sprays are the best antiseptics.

The Resection of the Liver.—AUVRAY (*Rev. de Chir.*, 1897, No. 4) has conducted a study of the possibilities of resection of the liver in the human being on animals that leads him to conclude that the procedure is applicable, in the living, to the ablation of tumors and cysts situated in the parenchyma of the liver, so long as they are not placed too deeply in the abdomen; that is, if they, for example, are situated in the left lobe or on the costal margins.

The method consists in passing through and through the parenchyma of the liver, outside of the line of resection, two interlooped sutures, which are so arranged that the various loops formed by each include and ligate the vessels of definite portions of the parenchyma in a continuous series.

Osteoplastic Closure of Bone-cavities.—OLIER (*Rev. de Chir.*, 1897, No. 4) speaks of the great difficulty experienced in the healing of wounds and the filling in of cavities produced by the removal of diseased spongy tissues from bones, especially the long bones, and, after studying the divers phases of the question, comes to the following conclusions:

1. Cavities in bones, whether abnormal, accidental, pathological, or following operations, fill up slowly, especially when they are deep, on account of the rigidity of their walls, which do not retract.. They cannot be filled by the granulations of medullary origin which form on their walls, at least when the subjects are not young and in a plastic condition. In tuberculous cases the difficulty is always very great.

2. In the long bones, as the tibia, after removal of the medullary substance, the repair is so tedious that it seems almost impossible to heal them. Of the short bones, the calcaneum is especially exposed to this slow repair where it has been reduced to but a thin wall.

3. The method to employ in healing these cavities is to remove or make movable one of the walls to permit, in the first case, the periosteum to reach the opposite wall, and, in the second, to place the osseous walls in contact.

4. When one of the bony walls is removed, the corresponding periosteum and soft tissues which cover it are pressed into the centre of the cavity and come in contact with the opposite wall. The strength of the bone is momentarily decreased by this procedure, but the formation of osseous tissue on the inner surface of the periosteum finally gives the necessary solidity. This is particularly true in cases of hypertrophy of the bone from osteomyelitis.

5. Outside of these cases, where hypertrophy has taken place it is better not to sacrifice the bone, but to form a flap which shall, according to the case, approach or be fixed to the opposite wall by metallic sutures. As soon as

the flap has been detached the cicatrization of the wound may be left to the granulations, for while they are without effect upon rigid walls, they act through the cicatricial contraction upon the movable wall. This release of one wall may be effected in many cases at the time the bone is operated upon and the cavity cleared. This precaution prevents the necessity of a secondary intervention.

6. The lateral flap is the easiest and most generally applicable. Many forms of flaps may be required, as cases differ. One may have recourse to flaps taken from other parts of the same bone or from neighboring bones, and changed by gliding or by partial torsion of the periosteal pedicle. This flap should always remain connected with the bone from which it was removed by periosteum and the soft parts, and as well nourished as possible to lessen the chances of necrosis of the flap. Where the bone is superficial the flap should be, as far as possible, osteocutaneous.

7. Bone-grafts, homoplastic or heteroplastic, may be employed, but only exceptionally, where the means to repair the loss of bone-substance cannot be found in the bone itself or any of the adjacent bones.

8. In certain cases where the cavities are intraosseous, with very thin walls in an atonic condition, it is best to proceed immediately to a resection of their entire thickness or to the total ablation of the bone, if it is a bone which is but of secondary importance to the solidity of the part.

The Cure of Tuberculous Peritonitis by Laparotomy.—A carefully conducted series of experiments for the purpose of studying the retrograde histological changes accompanying tuberculous peritonitis and laparotomy for its cure leads GATTI (*Archiv für klin. Chir.*, Band liii. Heft 4) to the following conclusions:

There are three stages observable in guinea-pigs, rabbits, and dogs—the fibrous, the præ-caseating, and the caseating—corresponding to the miliary, diffuse, and nodular forms of tuberculous peritonitis.

The operative procedure is without effect when it is undertaken too early—*i.e.*, before the tubercle has reached its complete histological development. If the operation is performed in a case of advanced disease, it may have no action upon the local disease, or, if it does, the animal may die from some other manifestation which has not been influenced by the interference.

A second laparotomy may be effective where the first is not, showing that it is often advisable to repeat the operation.

The simple laparotomy produces a bettering in the majority of cases, very frequently a macroscopic, and in a certain number of cases a histological, cure of the tuberculous peritonitis. This shows that a macroscopic, histologic, and biologic cure of tuberculous peritonitis is possible.

The histological healing takes place some months after the clinical recovery and the disappearance of macroscopic evidences. The complete healing-process extends over a period varying from six to eight months.

Fibrous tuberculosis disappears, both macro- and microscopically, after a simple laparotomy. The caseous variety is very much less influenced by operation. Often the development is slower; often retarded and made harmless. A disappearance of the caseating masses by resorption does not

take place. If there are present both forms, the fibrous will be healed while the caseating remains.

The healing of fibrous tuberculosis of the peritoneum by laparotomy is not due to an inflammatory reaction and development of connective tissue, but it is produced by a change in the epithelioid cells—a slow degeneration—which are then resorbed, while at the same time the normal cells and bacteria disappear, so that there finally remains nothing but the pre-existing connective tissue and its bloodvessels.

In caseous tuberculosis the connective-tissue capsule presents the same appearance both before and after operation, and is not thickened. The central caseous mass is thickened and may finally become entirely calcified.

The operation produces a change by which the tuberculous bacteria are killed or weakened or at least hindered from further development.

The protein of the killed or injured bacteria starts a slow degeneration of the epithelioid cells, which forms the basis of the histological degeneration of the tubercle.

The healing of tubercular peritonitis by a simple laparotomy may therefore be explained by the fact that as a result of the operation there is a fairly profuse serous exudate into the peritoneal cavity, which injures or destroys the tubercle bacilli and, under certain conditions, leads to healing through a retrograde metamorphosis.

The Technique of Suprapubic Lithotomy.—The feasibility of immediate suture after the removal of stones by the suprapubic method is discussed at length by STERLIN (*Deut. Zeitsch. für. Chir.*, Band xliv. Heft 3 u. 4), who comes to the conclusion that of cases in which this method of operating is indicated there are very few in which it is possible to secure primary union after immediate suture, and that the best method to pursue is that which he describes, as follows:

The patient is prepared by rendering the bladder so far as possible aseptic, and by two days of purgation. After narcosis the bladder is washed out repeatedly by a 3 per cent. solution of salicylic acid which is slightly warm. There should be great care exercised not to overfill the bladder and cause weakening of its muscular structure by that means. A two- to three-inch incision is made in the median line from the pubes upward, the recti muscles separated, and the left finger inserted, loosening the præ-peritoneal adipose tissue and gradually drawing the fold of perineum upward, as far as is necessary for the extraction of the stone. When possible the finger is allowed to remain while two silk sutures are inserted through the bladder-wall with a Reverdin needle and the bladder opened between them. The stone is now removed in the ordinary manner.

The next step is more difficult. He passes one or two sutures through the bladder-wall, the muscles, and the skin, thus forming a ventrofixation of the bladder. A soft catheter is now passed into the bladder, its tip cut off, a fenestrated drainage-tube passed over the tip, which is then drawn down into the neck of the bladder, while the drainage-tube is fixed by a safety-pin to the margin of the abdominal wound. There is thus secured ample drainage in both directions and a means of washing out the bladder. This washing is carried out twice daily, for the first two weeks, with the salicylic acid

solution mentioned above. The muscles, faciæ, and skin above the bladder-wound are united by silk sutures, and frequently from the pubes to the drainage-tube as well. The antiseptic dressing is changed twice a day at each washing out of the bladder. The patient should lie upon his side with the head of the bed raised. The fixation-sutures are removed at the end of two or three days and the drainage-tube one or two days later. The catheter remains in, on the average, one week. After its removal the patient has a bath daily, and a second retention-catheter is inserted and allowed to remain till no urine flows from the wound and the patient urinates himself. The washing out of the bladder through the catheter which is inserted late must be done with great care, in order not to rupture the union obtained in the abdomen.

The Antiseptic Value of Iodoform.—The results obtained by LOMRY (*Archiv für klin. Chir.*, 1896, Band xxxv. Heft 4) in a series of experiments conducted upon animals and by bacteriological cultures in natural fluids are in marked contrast to those obtained of late by bacteriologists, and tend to prove that the clinical observation of the value of iodoform as an antiseptic is fully justified, while the experimental bacteriological investigations which have been previously made were of little value.

The results which he obtained are the following:

1. When wounds in dogs or rabbits, which have been previously infected with the staphylococcus or streptococcus pyogenes, are treated the one with iodoform and the other without, it will be seen that the iodoform exerts a beneficial influence upon the wound. It has greater vitality and a better appearance. The leucocytes are at least as numerous and more active, the amount of secretion is decreased, and cicatrization is more rapid.

2. Credit cannot be given to the bacteriologists who declare that iodoform has no antiseptic action. They have attained their results by the use of culture-media which did not dissolve the iodoform. When natural media are used for tests *in vitro* an active antiseptic action is seen.

3. Iodoform decreases the virulence of the staphylococcus and streptococcus pyogenes.

4. The iodoform neutralizes or counteracts the microbic toxins, but this neutralization or counteraction is only partial.

5. The iodoform does not hinder, even to a slight extent, the amœboid movement and phagocytic action of the white corpuscles; but, on the other hand, stimulates their amœboid movement.

Exarticulation of One-half of the Pelvis for Extensive Caries following Coxitis.—In some cases of coxitis the disease extends far beyond the area of the hip-joint before it comes to operation, and in those cases where not alone the acetabulum but the bones which go to form the innominate are involved, the only method of preventing the spread of the disease, and certain death either by phthisis or amyloid disease, is a complete resection of the bones involved. WOLFF (*Cent. für Chir*, February 20, 1897) points this out most clearly, illustrating his subject by the report of a case in which the patient, forty-five years of age, was the subject of an extensive necrosis resulting

from a tubercular coxitis. There was marked amyloid degeneration, with a large amount of albumin in the urine.

Recovery and healing could only result from a rapid and complete cessation of suppuration. The only method by which this result could be attained was a resection of the diseased half of the pelvis. Five days after the operation the albumin disappeared from the urine, and the patient made a complete recovery in a month.

In operating the steps to be pursued are the tying of the external iliac artery and the hypogastric artery, thus preventing the loss of blood in a weak patient and hemorrhage in a diseased area where hæmostasis would be profuse and extremely difficult to deal with.

The next step is a typical resection of the joint, with a final exarticulation and resection of the half of the pelvis adjoining. The use of the chisel is to be avoided, as it increases the shock ; the bones should be sawn. The horizontal ramus of the pubic bone is resected at the inner border of the obturator foramen, as also the lower outer border of the ischium. The ileopsoas muscle is dissected from the inner surface of the innominate and the muscles attached to its superior border resected. The resected half of the pelvis is then disarticulated at the sacroiliac synchondrosis. The portion of the pelvis remaining is seldom diseased, and after careful curetting of the soft parts the muscles are united by buried catgut sutures in such a manner as to preclude hernia. The skin is then sutured after drainage has been secured and a compressing-dressing applied.

A New Instrument for Trephining.—DAHLGREN (*Cent. für Chir.*, 1896, No. 10) describes an ingenious instrument which he has devised, which has the essentials of simplicity and efficiency combined. After a small trephine-opening has been made and the dura mater pushed inward with a probe, it cuts a linear channel through the skull in any direction the operator may desire. It is especially adapted to the formation of the skin-periosteum bone-flap employed in Wagner's method of cranial resection.

The instrument consists of a bone-forceps whose jaws do not cross, but separate beyond the point of articulation. One arm carries a sharp hook which articulates at the extremity of the jaw and passes through between the two cutting-prongs of the other jaw. The bone is grasped and cut through by the hook while pressed against the outer surface of the other jaw. By means of a set-screw the instrument can be accurately adjusted to the thickness of the bone to be cut through.

The Treatment of Carcinoma by the Erysipelas-serum of Emmerich and Scholl.—In addition to the varied expressions of distrust that accompanied the publication of this method of treatment the following results obtained in Russia by THIELE (*Annalen der Russ. Chir.*, 1896, Heft 3) are noteworthy: (1) The complete cure by means of the serum was not observed. (2) The injections are not dangerous, but are not harmless (they produce headache, weakness, increased temperature, etc.). (3) The cancerous nodules become temporarily swollen, the growth of the tumor is checked for a time. (4) In inoperable cancers the serum can be used, as it decreases the pain and thereby the amount of narcotics required. (5) The systematic study of this method applied to early growths is much needed to demonstrate its true value.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

EDWARD JACKSON, A.M., M.D.,

PROFESSOR OF DISEASES OF THE EYE IN THE PHILADELPHIA POLYCLINIC; SURGEON TO WILLS EYE HOSPITAL, ETC.

ASSISTED BY

T. B. SCHNEIDEMAN, A.M., M.D.,

ADJUNCT PROFESSOR OF DISEASES OF THE EYE IN THE PHILADELPHIA POLYCLINIC; ASSISTANT SURGEON TO WILLS EYE HOSPITAL, ETC.

Permanent Central Scotoma Caused by Looking at the Sun.—E. TREACHER COLLINS (London) reports the case of a woman, aged forty-nine years, who, after gazing at the sun for five or ten minutes, noticed, upon turning away, yellow disks before the eye. The yellow changed to blue in a shaded room and looking at a white surface. These disks were only faintly visible the next day and entirely gone the day after; but there seemed to be cobwebs in front of her eyes, and by the seventh day the sight in the left eye was gone. At the end of three or four weeks the right eye had entirely recovered. Six months later she was found to present normal ophthalmoscopic appearances, except that the left disk was paler than the right throughout, and its outer half markedly white; and each macula presented small shining white spots. She had full vision with the right eye, and with the left could only count fingers at three feet. The fields were of normal size, and the left presented a central scotóma, especially for red, and the field for green was almost abolished, while yellow was called white in all parts of the field, but blue could be distinguished except at the centre. The case is remarkable for the permanence of the scotoma and the occurrence of noticeable optic atrophy.—*Royal London Oph. Hosp. Rep.*, vol. xiv. p. 374.

Spontaneous Hemorrhages into the Vitreous.—PANAS (Paris) claims that infection is at the bottom of all such cases. The source of the infection may reside in a general dyscrasia like syphilis, gout, or alcoholism, and perhaps gonorrhœa, or may depend upon a local focus situated, for example, in the nasal fossa or the intestinal canal. Venous thrombosis most likely precedes the hemorrhages in many instances. This has been shown to be the mechanism in leucæmic retinitis and certain ocular affections due to malaria. The prognosis is extremely unfavorable, not only as regards the affected eye, but also from the great danger of a like occurrence in the other.

Treatment should be directed to the underlying cause: disinfection of the nose, if it is diseased, abstinence from alcohol where necessary, antisyphilitic remedies when this disease is present. In the latter case, iodides are not of much service. Mercury is the sovereign drug, especially by hypodermatic injection. This remedy is indicated as a general antiseptic, even in the absence of specific disease. Arsenic is also useful.—*La Presse Médicale*, November 27, 1897.

Ophthalmia Caused by Caterpillars.—NATANSON (Moscow) calls attention to this animal as a cause of disease of the skin, mucous membranes, and eyes, either directly by contact (in a few cases), or indirectly through diffusion of its hairs giving rise to widespread endemics. In both cases the hairs are the noxious agent through their toxic and mechanical action. The toxin is formic acid.

The ocular affection occurs under two forms—mild and severe. The endemics are of the former variety, and run their course as bilateral conjunctivitis, lasting from one to two weeks. The severe and much rarer form is caused by direct injury of the eye by contact with the animal itself. The conjunctiva, cornea, and anterior portion of the uveal tract are the parts affected. Small nodules quite analogous to miliary tubercles and containing hairs are usually present in those structures.—*St. Petersburg. med. Wochenschrift*, xxii. No. 12.

Glaucoma after Cataract Extraction.—S. C. AYRES (Cincinnati) reports five cases (six eyes) of glaucoma following extraction of cataract. In five of these eyes extraction had been done with a liberal iridectomy; in the other eye extraction was practised after preliminary iridectomy. The first case had undergone extraction from the left eye in 1867 and the right in 1875. In 1895 the latter developed symptoms of glaucoma. The vision in the left eye had failed eight years before. In both there was contraction of the visual field, deep excavation of the disks, and vision reduced to counting fingers. The left eye showed a cystoid cicatrix at each end of the wound.

In the second case glaucoma supervened two years after the extraction, and the disk became deeply cupped and atrophied, with vision quite abolished.

In the third case, in which an upward preliminary iridectomy had been performed, and vision of 20/XL had been obtained after extraction, pain was noticed at the end of seven weeks, and some weeks later the glaucoma became acute, iridectomy downward gave no relief, and the eye was subsequently enucleated.

In the other two cases acute glaucoma developed on the sixth and twelfth days after the extraction. Both were cured by evacuation of the aqueous and the use of eserine. In one of them atropin had been used; and in the other glaucoma developed after exposure in a snowstorm.—*Archives of Ophthalmology*, vol. xxvi. No. 2.

Interstitial Keratitis in Acquired Syphilis.—TROUSSEAU (Paris) reports eleven cases of interstitial keratitis in subjects of acquired syphilis, and refers to certain points of likeness and difference between this condition and that due to hereditary lues. As with the latter, the majority (8) occurred in females; but, unlike the inherited, the acquired disease is much milder, affects only one eye, and heals much sooner and with little or no tendency to relapses. Treatment by mercury is very effective.

Trousseau attributes the milder course of the disease to the superior resisting-power of the adult tissues.—*La Presse Médicale*, 1897, No. 40.

Ophthalmoplegic Migraine.—BOUCHAUD (Paris) reviews the scanty literature of this rare disease and reports a case. The affection is characterized by

a series of periodic crises of unilateral cephalalgia, with paralysis of the third nerve of the same side; all the branches are affected, but not always to the same degree. Finally the paralysis becomes permanent.—*La Presse Médicale*, 1897, No. 34.

Glaucoma and the Curative Action of Iridectomy.—ABADIE (Paris) believes the cause of glaucoma to consist in derangement of the constrictor and dilator nerves of the sympathetic system. These form a plexus in the iris, excision of which inhibits superaction of the dilator fibres.

He bases his view upon the fact that excision of the pupillary or ciliary portion of the iris is ineffective in acute glaucoma, but the intermediate zone which contains the ganglia has to be removed if the operation is to be curative.—*La Médecine Moderne*, 8 Annee, No. 38.

Sudden Loss of Vision of Circulatory Origin.—J. B. BARRETT (Melbourne) reports two cases of this affection, with a very thorough study of the literature of the subject.

His first case is that of a young medical man, who noticed, a few days before examination, that a sudden mist passed before the left eye; he was at the time standing conversing in a room. The vision failed so completely that he was unable to distinguish light from darkness; for five minutes there was complete blindness. The ceiling of the room became visible, and gradually the field cleared from above downward, until he could see all portions above the level of his own head. After a short stoppage at this point the field cleared completely, and no noticeable alteration remained. He has never had another similar attack, and was apparently in perfect health.

The second patient, a lady aged thirty-three years, noticed, while writing, that the vision of the right eye was affected, and on covering the left eye found that the right was totally blind. The blindness lasted for about fifteen minutes, and then the lower half of the field of vision gradually cleared. The upper half of the field has remained blind ever since. The defect gave a sensation of inability to raise the uper lid. The patient has suffered from shorter attacks of complete blindness—she thinks chiefly in the right eye—on about seven occasions during the last ten years. On previous occasions the blindness lasted less than one minute and left behind it no permanent affection of vision.

The eye was examined the day after the attack, and then exhibited the typical appearance of embolism of the inferior retinal artery.

From his study of recorded cases Barrett believes that spasm of the retinal arteries can cause blindness. How long the retinal circulation can be arrested without permanent destruction of vision cannot be determined, although in one case Noyes secured complete recovery by the use of nitrite of amyl, twenty-four hours after the sight was lost. The condition of the vessels when the diseased eye has been examined microscopically has been that of an embolism or a thrombosis, although in three cases out of twelve no obstruction could be discovered.

Barrett concludes that sudden temporary loss of vision may be produced by: (1) Spasm of the retinal arteries. (2) Complete embolism of the retinal artery. (3) Partial embolism of the retinal artery, probably combined with

spasm. (4) Narrowing of the retinal arteries from arterial disease and temporary heart-failure. Several of these causes may co-operate with one another.

Sudden permanent blindness may be produced by: (1) Spasm of the retinal vessels. (2) Complete embolism. (3) Partial embolism, with temporary spasm of the arteries. (4) Thrombus, with or without spasm. The conjunction of complete temporary loss of vision in both eyes, with permanent partial loss in one, seems almost to eliminate the possiblity of causation of embolus.—*Intercolonial Medical Journal of Australasia*, vol. ii. No. 3.

Quinine-amaurosis without Pallor of the Optic Disks. — E. GRUENING (New York) reports two cases. The essential features of quinine-amaurosis are the sudden occurrence of deafness and blindness, the return of the function of hearing within twenty-four hours, the restoration of central vision after days, weeks, or months, permanent abolition of peripheric vision, ischæmia of the retinal arteries and veins, and the absence of inflammatory changes in the interior of the eye.

Quinine-intoxication associated with transitory amaurosis, however, occurs at times in milder forms, showing a less pronounced contraction of the retinal arteries and veins and a decided fulness of the capillaries of the optic disk as a permanent condition.

One of his patients was a woman, aged fifty years, who had taken thirty grains of quinine in one dose in the evening, and awoke next morning totally blind and deaf. Hearing and sight returned; central vision and color-perception became normal in a few days, but the field of vision remained permanently contracted.

The second patient was a man, aged seventy-three years, who took 150 grains of quinine in three days and suddenly became deaf and blind. Hearing and sight returned in thirty-six hours, but the visual field remained greatly contracted.

In both cases the optic disks remained red rather than pale, and the retinal vessels were but little altered.—*New York Eye and Ear Infirmary Reports*, 1897.

Operation for Divergent Strabismus.—CHARLES BELL TAYLOR (Nottingham, Eng.) corrects divergent strabismus by the following method: He hooks up the tendon of the internal rectus muscle and divides all its connections with the eyeball, then pulls it well forward, and, by two horizontal incisions made with scissors, converts it into a narrowish strip. He then transfixes the base of this strip as far back in the orbit as possible with a large curved needle connected with a thread armed at both ends. Before drawing the thread through he cuts off as much of the tendon as may be considered necessary. With the remaining needle he pierces a small portion of the sclerotic about the site of the insertion of the inferior rectus tendon, and having thus secured control of the eyeball, turns it inward to any required extent by simply tying the thread. The only portion of the procedure which presents any difficulty is the piercing of the sclerotic. To overcome this he uses needles of the same calibre throughout, and as sharp as possible, and makes the insertion of the thread the first step of the operation.—*Medical Press and Circular*, March 10, 1897.

Cicatricial Contraction of the Orbital Tissue after Enucleation.—Dr. G. C. Harlan (Philadelphia) calls attention to a new method of dealing with this difficulty. In a case where there were a number of cicatricial bands extending from the lids to the bottom of the conjunctival sac, which obliterated the sulcus and made it absolutely impossible to wear even the smallest artificial eye, the attempt was made to form an artificial sulcus by passing a thick lead wire around the bottom of the cavity through the contracting bands, and allowing it to remain in position until the walls of the sinus it had formed had completely cicatrized or "skinned over." The patient wore the wire without inconvenience for six or eight weeks, when it was removed by incising the tissue in front of it, and a leaden shell was inserted whose edges rested in the groove where the wire had been. Later, an artificial eye was substituted for the leaden shell, and has been worn since without inconvenience.—*Ophthalmic Record*, April, 1897.

The Treatment of Lachrymal Obstruction by Styles.—R. Shalders Miller (Windsor, Eng.) treats all organic strictures of the lachrymal passages by means of styles, in most cases permanently retained. Except for temporary wear, they should be made of pure platinum wire, about 25 gauge, and he makes them himself for each case. The shape, direction, and length of the nasal duct being variable, special adaptation is the key to success in the use of styles, which consist of a vertical stem, with a short arm bent at right-angles to the stem. Usually this arm is made double by recurving the wire back to the stem, so that the arm consists of two thicknesses of wire, parallel and quite close together, the recurved part lying vertically beneath the other.

The tubular styles of the instrument-makers are worse than useless; they soon become choked with mucus and dust. Also, in the case of grooved styles, the necessarily thin edges of the furrow cause irritation and the production of a granulation which stops the way. The stem of a style should not be straight, except in the case of a very short duct, but somewhat concave outward.—*British Medical Journal*, March 13, 1897.

Sympathetic Inflammation of the Eye.—E. Donaldson (Londonderry) reports a case of sympathetic ophthalmia occurring after enucleation of the exciting eye for rupture of the sclerotic, the conjunctiva not having been ruptured. The disease continued active more than five years after its onset. The progress of the case was very variable; sometimes the eye was quiet, then again it became painful, vision grew dim, and ciliary congestion returned. The evil effect produced by catarrhal ophthalmia occurring in an eye already affected with sympathetic ophthalmia was noted. The microscope showed a plastic cyclitis in the enucleated eye. Vision equalling 4/60 still remained in the sympathizing eye.—*Ophthalmic Review*, February, 1897.

Meningitis following Excision of the Eyeball for Panophthalmitis.—C. Devereaux Marshall (London) states that between the years 1861 and 1896 there have been 6580 eyes excised at Moorfields Hospital, among which 8 fatal cases have occurred. He reports 5 of these cases with autopsies and

microscopical examinations, and from an extended study of the subject concludes:

1. Meningitis may be present for a certain time without there being sufficient symptoms to enable one to diagnosticate the disease.

2. Meningitis has been known to follow other operations besides the excision of suppurating eyes, and cases are also recorded in which the excision of an eye which was not suppurating has been followed by death from meningitis.

3. The changes seen in many cases indicate that the disease is of older standing than the symptoms would appear to indicate.

4. Infection may occur at any time from an eye which is suppurating, and the longer the pus is shut up in the eye the greater is the risk and the greater will be the absorption of products of suppuration.

5. There are two ways in which meningitis may arise: (*a*) by direct extension along the optic nerve and structures passing through the sphenoidal fissure; (*b*) by infective material being carried along the vessels.

6. The sooner the pus is got rid of the better; and if it is thought not desirable to excise the eye, it should be at once opened, the contents completely removed, the sclerotic thoroughly scrubbed out, and both it and the surrounding parts rendered aseptic. Owing to there being no clear cornea to injure, a far stronger antiseptic may be employed than would be possible if a seeing eye were present.

7. As the products of putrefaction may have soaked into the sclerotic and infected the surrounding parts, it is far better to remove it; good drainage is then insured, and every piece of useless and suppurating tissue is removed. —*Royal Lond. Ophthal. Hosp. Rep.*, vol. xiv. p 305.

DERMATOLOGY.

UNDER THE CHARGE OF

LOUIS A. DUHRING, M.D.,

PROFESSOR OF DERMATOLOGY IN THE UNIVERSITY OF PENNSYLVANIA;

AND

MILTON B. HARTZELL, M.D.,

INSTRUCTOR IN DERMATOLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

On Ringworm: an Inquiry into the Plurality of its Fungi.—T. COLCOTT FOX and FRANK R. BLAXALL have collected and published in brochure-form (London, 1897, pp. 65) their valuable papers which have appeared in the *British Journal of Dermatology* during the past year. The subject is one of great interest, especially since the elaborate and scientific investigations of Sabouraud in Paris. Fox and Blaxall's conclusions, based upon a clinical, microscopical, and cultural investigation of more than 400 consecutive cases of ringworm of the scalp, of the beard, and of the glabrous skin, cor-

respond generally with those reached by Sabouraud. They can distinguish clinically, microscopically, and by cultures the group separated by Gruby and Sabouraud and associated with the growth of *microspora;* also another group associated with the growth of *trichophyta.* The last group is divisible into two secondary groups, denominated *endothrix trichophyta* and *ectothrix* or *endoectothrix trichophyta.* In London the *microspora* are probably the cause of 80 to 90 per cent. of cases of ringworm of the scalp; the *endothrix,* of nearly 4 per cent.; and the *ectothrix* or *endoectothrix,* of the remainder. The cases of beard-ringworm appear to belong to the last group, and the majority of cases of ringworm of glabrous skin, exclusive of those associated with lesions of the scalp, are caused by *trichophyta.* Fox and Blaxall have not met with an example corresponding to the "peladoid" (or alopecia areata) form described by Sabouraud. It may be well to state that Sabouraud holds that in Paris the ringworms which he met with in human beings comprised two affections caused by fungi as distinct from one another, clinically, morphologically, culturally, and botanically, as those of tinea versicolor or tinea favosa, from either of them. The more frequent one is ringworm with small spores, the special fungus described by Gruby, in 1843, under the name of *microsporon Audouini.* The other affection comprises the ringworms with comparatively large spores, the true *trichophytosis* of all sites, caused by various species of *trichophyton* fungus, belonging to a family distinct from the *microsporon.* These *trichophytons* are divisible into two great groups, the one apparently of exclusively human origin, characterized by a marked tendency to affect only the interior of the hair when that structure is invaded, and hence denominated *trichophyton megalosporon endothrix;* the other apparently of animal origin, and locating itself chiefly between the wall of the follicle and the hair as well as entering the shaft, and hence called *trichophyton megalosporon ectothrix* or *endoectothrix.* The *endothrix* fungus causes about 35 to 40 per cent. of all scalp-ringworm in Paris and about 72 per cent. of the scalp-ringworms with large spores. The authors call attention to the fact that ringworm fungi (also the fungi of tinea favosa) have a very unequal distribution in European countries. A lesson is to be learned from the admirable work done by Sabouraud, Béclère, the authors, and others in ringworm, and that is, that one should be cautious in criticising the results obtained by observers in different countries. Numerous photomicrographs accompany the article.

The Etiology of Trichorrhexis Nodosa —BRUHNS (*Archiv für Dermatologie und Syphilis,* Bd. xxxviii. Heft 1), who has studied trichorrhexis nodosa as it occurs in Berne, concludes a paper on this subject as follows: trichorrhexis nodosa capilitii is very frequently observed in women in Berne and vicinity. The bacterial investigations of the disease and the inoculations undertaken therewith have not proved that it is produced by a specific bacterium. The author failed to find a bacterium as the cause in six cases investigated by him with this end in view; and inoculation upon the hairs of other individuals failed to produce any result. On the contrary, some facts are very much against a bacterial etiology—as the occurrence of trichorrhexis periodically in connection with attacks of epilepsy, as reported by Raüber. On the other hand, it is improbable that it varies only through mechanical causes;

there must be some predisposition to the affection, brought about probably by disturbance of nutrition.

Myoma Multiplex of the Skin.—CROCKER reports (*British Journal of Dermatology*, January and February, 1897) an example of this rare affection, it being the eleventh recorded case. The patient was a man, forty-three years of age. Eighteen years before coming under the reporter's observation a single tumor appeared on the lower jaw of the left side. Two years later another appeared about one inch from the first one; and since that time new ones gradually appeared until they formed a patch about an inch by an inch and a half in area, containing twenty-seven tumors varying in size from a hempseed to a large bean. They were brownish-red in color, firm to the touch, and entirely painless. The treatment consisted in the excision of the entire patch.

The author presents likewise an abstract of all the cases hitherto reported. From an analysis of these it appears that in seven the affection occurred in females; in four in males. The age at which the disease first made its appearance varied from infancy to sixty years; the size of the tumors varied from a millet-seed to a hazelnut; the number was very variable, and while in some cases they occurred singly, in most there was a marked tendency to form groups. The color was brownish-red or some shade of red, and the skin over the tumors was always normal. A microscopical examination of the author's case showed that the epidermis was unaffected, the growth being seated for the most part in the deep part of the corium, leaving the papillary body free in some sections, but going up to the papillæ in others. The small tumors were seen to be situated about the hair-follicle, the follicle being surrounded with a thick layer of smooth muscular fibres. These could also be seen in some sections about the sebaceous and sweat-follicles. Bloodvessels were not numerous, but where present showed evident thickening of the muscular coat.

OBSTETRICS.

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.,

PROFESSOR OF OBSTETRICS AND DISEASES OF INFANCY IN THE PHILADELPHIA POLYCLINIC; CLINICAL PROFESSOR OF OBSTETRICS IN THE JEFFERSON MEDICAL COLLEGE; CLINICAL PROFESSOR OF DISEASES OF CHILDREN IN THE WOMAN'S MEDICAL COLLEGE; VISITING OBSTETRICIAN TO THE PHILADELPHIA HOSPITAL, ETC.

Shock in Obstetric Surgery.—In the *American Gynecological and Obstetrical Journal*, April, 1897, COE, of New York, contributes an interesting paper upon this topic. He cites six cases in which delivery by forceps and embryotomy was followed by shock which in four cases proved fatal. He adds a case of ruptured uterus in which the rupture was not detected at labor, in which the patient died after abdominal section. He also describes

a case of a patient who had aborted and suffered from severe shock after curetting. His list of cases closes with that of a patient who perished from shock accompanied by rapidly developed pulmonary œdema.

Coe has observed that this complication is most apt to occur in fat, neurotic women often in comfortable circumstances, and sometimes accustomed to luxury. Old primiparæ are especially exposed to this danger.

[The reviewer is in full accord with Dr. Coe in the warning which he expresses in this paper. No medical man is burdened with so heavy a responsibility or obliged to work in such unfavorable conditions as the obstetrician. All who practise obstetrics should unite in educating the community to acknowledge the fact that a difficult labor requires the care and assistance which are invariably employed in conducting abdominal sections. Unless such precautions be taken, the mortality-rate of difficult confinements in private houses must always remain a very high one.]

The Treatment of Fibroid Tumors Complicating Pregnancy and Labor.—At a recent meeting of the Obstetrical Society of France (*Gazette Hebdomadaire*, 1897, No. 34) KEIFFER read an extended paper upon this subject, in which he drew the following conclusions: hysterectomy in fibroid uteri in pregnant patients is demanded for four indications: (1) when, independently of pregnancy, the fibroid tumor would make hysterectomy justifiable; (2) when the fibroid occupies such a position that labor would be impossible; (3) when the tumor is degenerating, or suppurating, and when a retained placenta complicates the case; (4) hysterectomy should be performed in a case of labor complicated by fibroid tumor of the uterus after the child has been extracted by Cæsarean section.

Habitual Abortion.—CHARPENTIER contributed a paper upon this subject at the recent meeting of the Obstetrical Society of France (*Gazette Hebdomadaire*, No. 34, 1897), in which he stated that four classes of patients are especially liable to repeated abortion. The first of these have some malformation of the uterus which prevents its normal development during pregnancy. Frequent contractions of the womb follow conception, and abortion finally occurs about the second or third month. A second class is composed of those women in whom the womb is abnormally displaced. They sometimes go to five or five and a half months. A third class of patients exhibits intense congestion about the neck and body of the uterus during pregnancy. These women sometimes bleed during pregnancy at the usual time for menstruation. Others suffer greatly from hemorrhoids. A fourth class especially liable to abortion are those in whom a tumor of the uterus, either in the neck or body, is present; such are cases of fibroid tumor or sarcoma. It is evident that the treatment of these patients should be addressed to remedying the existing condition which causes the abortion.

The Temperature of Breast-milk.—SMESTER, in the *Maladies de l'Enfance*, 1897, No. 15, has made experiments upon the temperature of milk as it is taken from the breast of the mother. He used a form of breast-pump containing a thermometer, so that the temperature of milk could be measured without admitting the external air. He finds that as the milk enters

the child's mouth its temperature is always below 98° F. It usually varies from 96.5° to 97° F. It is evident that milk given to children should not be heated above these temperatures, and Smester claims that infants naturally decline milk warmed to any degree.

Investigation into the Percentage of Hæmoglobin and the Number of Red and White Corpuscles during Pregnancy and the Puerperal State.—In the *Archiv für Gynäkologie*, Band liii, Heft 2, WILD gives the results of his observations upon this subject in the clinic at Zurich. He finds that during the last weeks of pregnancy there is a slight increase in hæmoglobin in the blood of the mother, with a slight decrease in the number of red blood-cells and a striking increase in the number of white cells. During labor the hæmoglobin and red cells are diminished through loss of blood. During the puerperal period the patient gains in hæmoglobin and red blood-cells. The white cells are most abundant immediately after labor and diminish somewhat during the puerperal state. Lactation exercises a favorable influence.

The Presence of Bacteria in the Milk of Nursing-mothers, and its Relation to Mastitis.—In the *Archiv für Gynäkologie*, 1897, Band liii, Heft 2, KÖSTLIN reports the result of a series of investigations in the clinic at Halle on this subject. He finds that in the majority of cases the secretions of the breast in pregnant and puerperal women and even in the newborn contain bacteria. In pregnant women this was true in 86 per cent. of patients examined, in puerperal women in 91 per cent., and in newborn infants in 75 per cent. With very few exceptions these germs were staphylococci, and especially the *staphylococcus albus*. In these cases no point of entry for these germs was found nor any circumstances explaining their presence. They must have entered from without through the nipples, and especially from the areola about the nipples. It has not yet been possible to recognize them in the blood-current. The presence of these germs was harmless to mother and child. The view formerly held, that mastitis depends upon micro-organisms, is still correct. The infection in mastitis comes from without, through a lesion in the skin communicating with the lymph-channels, and spreads itself in different ways in the case of different germs. The ordinary form of mastitis results from invasion of staphylococci, especially the *staphylococcus aureus*. The less common forms of mastitis, such as pseudo-erysipelas and retromammary abscess, are caused by streptococci. All means of study of these cases point to common conclusions; mixed infections are possible, and not uncommon; a mastitis caused by metatastic infection through the blood-current has not as yet been clearly proved.

Extravasation of Blood in the Vertebral Canal in Newborn Infants.—In the *Archiv für Gynäkologie*, 1897, Band liii, Heft 2, SCHAEFFER reports some very interesting studies upon this subject made in the Heidelberg clinic. He finds that in 100 autopsies upon newborn infants that in one in every ten extravasated blood was found in the vertebral canal, while cerebral extravasations were as frequent as two in every ten. In seventeen cases of vertebral bleeding 41 per cent. followed the use of forceps or extraction in

breech presentation. Some of the children perished during birth; others at varying periods afterward; 24 per cent. of these had other birth-lesions; these were principally lacerations or injuries of the cerebral mass. In the whole number of children dying, 64 per cent. died of birth-injury; 29 per cent. perished of disease or injury resulting indirectly from birth.

It was observed that prematurely born children most often sustain birth-injury, while those born at term develop post-partum diseases. So far as regions of the spine were concerned, in which bleeding was most frequent, in three cases extravasation occurred in the medulla; in two cases in the cervical region; in three cases in the cervical and dorsal; and in two in the dorsal and lumbar. These were all in children who had been extracted in breech presentation. So far as prognosis is concerned, when bleeding occurred into the region of the medulla the child died. The result depended, of course, somewhat upon the amount of hemorrhage, although that did not seem a deciding factor. In asphyxiated children and in premature children any strong mechanical excitement, such as extraction, and above all Schultze's manipulation, can cause such hemorrhage. The prevention of this complication consists in warding off all infection during and after labor and in using those methods of delivery which offer least violence to the fœtus. It is especially noticeable that the cautious application of forceps gave excellent results.

GYNECOLOGY.

UNDER THE CHARGE OF

HENRY C. COE, M.D., M.R.C.S.,
OF NEW YORK.

Renewal of the Endometrium after Cauterization.—JUNG (*Centralblatt für Gynäkologie*, 1897, No. 18) contributes an interesting paper on this subject, based on microscopical studies of a uterus removed four months after thorough cauterization of its interior. The operation was performed for obstinate hemorrhage, which had not been controlled by curettement and application of chloride of zinc paste. An entire cast of the uterine cavity had been passed. On examination of the endometrium it was found that renewal of the epithelial layer had begun, though only over a small region. The new mucosa was in the same hyperplastic condition as before the cauterization. This proves that the endometrium "is a tissue possessing an inexhaustible power of regeneration," no matter how thoroughly it may be destroyed. Microscopical investigations by Werth show that young connective tissue is renewed within a few days after the traumatism from similar tissue in the muscularis. The former is only temporary, being replaced as early as the tenth day by normal stroma. The glands redevelop from remnants of pre-existing glands, the superficial epithelium from that lining the mouths of the newly formed glands. The growth of the new vessels keeps

pace with that of the regenerated mucosa, being complete even on the fifth day.

Bossi's conclusions were somewhat different. He removed the endometrium and a portion of the muscularis from the uteri of dogs, and found that regeneration was complete in three months. He observed that the glands developed from remains of old glands only at the border of the raw surface; in the centre of the wound the glands were formed by ingrowth of the new surface-epithelium into the subjacent connective tissue. The writer finds the conditions in his own specimen similar to those caused by experimental traumatism in Bossi's cases; the results were different, since after the cauterization regeneration was only partial, and the mucosa was renewed in six weeks, as shown by a return of the hemorrhages.

The writer concludes that the use of chloride of zinc within the uterine cavity for the relief of hemorrhage, in paste or strong solutions, is reprehensible. Although the destructive character of the escharotic is marked, it is uneven and cannot be controlled, while there is great danger of producing stenosis. The application should not be repeated within less than two weeks, in order that the raw surface may have time to heal. The use of a 50 per cent. solution, applied on cotton, is preferable to pencils or intrauterine injections.

Conception after Curettement of the Cancerous Cervix.—LEINZINGER (*Ibid.*) reports a case of this character, in which a patient was admitted to the hospital with inoperable carcinoma of the cervix. She was curetted on November 26, 1895, and was discharged improved. She remained well for ten months, when the operation was repeated, November 17, 1896; she became septic, and was again curetted; aborted with a four and a half months' fœtus, and died eleven days later. The writer emphasizes the following points: 1. The relief afforded by the curettement, extending over nearly a year. 2. The occurrence of pregnancy, which seemed to have no unfavorable effect upon the development of the disease. 3. The absence of all symptoms which would awaken the suspicion of existing pregnancy. (The enlargement of the uterus was supposed to be due to hydrometra.) 4. The profuse hemorrhage attending the third operation undoubtedly hastened the fatal termination. [We have been personally cognizant of several cases in which abortion followed curettement for advanced carcinoma of the cervix, pregnancy being unsuspected; in one instance of recurrence after high amputation we succeeded in carrying the woman to full term and a successful delivery, and were afterward able to extirpate the uterus, the patient being still in good health four years after the operation.—H. C. C.]

Vaginal Hysterectomy with the Use of Clamps.—AREND (*Ibid.*) repeats the usual arguments in favor of clamps *versus* ligatures. He claims that the pain caused by the former is not greater than when ligatures are used, and that the danger of hemorrhage is much less, though he always removes the instruments at the end of forty-eight hours, the gauze being entirely withdrawn on the fifth or sixth day.

[Too little stress is laid upon the extensive sloughing-process which attends the use of clamps, and requires most careful after-treatment to insure a rapid

and afebrile convalescence. The writer's experieence is too limited to allow of *ex cathedra* statements.—H. C. C.]

Massage in Trendelenburg's Posture.—BENTTNER (*Centralblatt für Gynäkologie*, 1897, No. 19) recommends this posture for the following reasons: 1. The oblique abdominal muscles are more relaxed. 2. The intestines fall toward the diaphragm, so that massage is easier in fat subjects. 3. Women feel less weary after manipulations in this posture. 4. The uterus and adherent adnexa are carried upward *en masse*, becoming more accessible to the external hand. The writer found that patients who had tried massage in both the dorsal and elevated postures preferred the latter.

Constipation in the Female.—PINCUS (*Archiv für Gynäkologie*, Band liii. Heft 3) has made an exhaustive study of the causes of chronic constipation in the female, which he believes is due not only to loss of tone in the muscular layer of the intestine, but to weakening of the abdominal and levatores ani muscles. In the treatment of this condition he relies upon massage, electricity, and gymnastic exercises, to the exclusion of laxatives and enemata. After a "preparation" of from one to six weeks, during which time the bowels are thoroughly emptied by high enemata, he affirms that the most obstinate case of constipation can be cured in from three to nine days. Abdominal massage must be practised in a scientific manner, hence only by a physician. A good substitute for massage is the systematic use for ten minutes, morning and evening, of a bag filled with stones or bits of iron, which is rolled about over the abdomen. Many interesting details are given.

Cancerous Degeneration of Dermoid Cysts.—THUMIM (*Ibid.*) reports a case of epithelioma developing on the inner lining of an ovarian dermoid. He has no explanation to offer with regard to the etiology. Malignant degeneration of dermoid may occur at any age, but is always unilateral. Metastases are local, ascites is rare.

The diagnosis is difficult. Unusually rapid growth of a dermoid, associated with progressive emaciation, ascites, and cachexia, might cause the suspicion that malignant degeneration had occurred.

The prognosis will depend upon the stage at which the tumor is removed. In the case reported, in which the disease was advanced, an early recurrence took place, and the patient died six months after the operation.

Hemorrhagic Endometritis in Virgins.—BLANC (*Loire médicale*, December 15, 1896) calls attention to the fact that, although menorrhagia in virgins has often been observed, the treatment has been usually simply medicinal, and therefore not curative. He recommends curettement as in cases of ordinary hyperplastic endometritis in married women. The following cases are cited:

Case I.—Girl, aged sixteen years, with menorrhagia of eighteen months' standing, beginning with her second menstrual period. So much blood was lost that death became imminent. Curettement was followed by relief for six months, when a second operation was necessary. There was no return of the hemorrhages after the lapse of six years.

Case II.—Menorrhagia of a year's standing in a girl, fifteen years of age,

with pelvic pains and marked anæmia. The curette removed a quantity of pale fungosities; the patient was cured.

Case III.—Hemorrhages recurring at intervals for six months in a child of fourteen. Cure by curettement.

Case IV.—Obstinate hemorrhages in a patient, aged fifteen years, which had continued for six months in spite of the administration of iron, ergot, hydrastis, and hot douches. An operation was refused and the patient eventually succumbed.

Congenital Prolapse of the Uterus.—BALLANTYNE and THOMSON (*American Journal of Obstetrics*, 1897, No. 2) report two cases, making eight which have been published. They call attention to the importance of distinguishing true prolapse from hypertrophic elongation of the cervix, although the two conditions may coexist. Congenital prolapse is commonly associated with spina bifida, prolapse of the rectum, and sometimes flat-foot. These infants usually die soon after birth, probably from causes attributable to the presence of the spina bifida. The prolapse may be partial or complete, but rarely gives rise to symptoms. The etiology is usually to be sought for in faulty innervation due to the spina bifida; but increase of the normal abdominal pressure, unusual size of the pelvis, and hypertrophy of the cervix or corpus uteri are doubtless contributing factors.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

EDWARD F. WILLOUGHBY, M.D.,

OF LONDON;

AND

CHARLES HARRINGTON, M.D.,

INSTRUCTOR IN MATERIA MEDICA AND HYGIENE, HARVARD MEDICAL SCHOOL.

Soaps as Disinfectants.—Much experimental work has been done within the past fifteen years to determine the place which soaps should occupy as disinfectants. In 1881 Koch showed that potash soap, 1 : 5000, would materially diminish the growth of anthrax-bacilli, and 1 : 1000 would prevent it completely; and that potash soap was eight times as powerful as potash itself. In 1885 Kuisl asserted that while it acted on anthrax, it was quite inert against other kinds of bacteria. Thus, typhoid bacilli would thrive in the presence of 2 per cent. of soft soap, as would also the spirillum of cholera. In 1890 Behring experimented with forty different soaps, and concluded that their disinfectant power was dependent on their alkalinity. In 1893 Nijland, experimenting with a soft soap containing 42.7 per cent. water, a medicated soap containing 7.3 per cent., and a hard washing-soap containing 14.5 per cent., found that when used in 0.24 per cent. solution the first would kill

cholera germs in ten minutes, the second in fifteen, and the third not completely in fifteen, but in 0.30 per cent. solution within one minute. Jolles, in 1893, tried five soaps, and proved them to be powerful agents; and in 1895, experimenting with another soap, which was almost neutral in reaction, containing but 0.041 per cent. free alkali, found that typhoid bouillon was sterilized by 1 per cent. within twelve hours, by 3 per cent. within two hours, and by 6 per cent. within fifteen minutes, at 4° to 8° Centigrade. At 18° C. the action was weaker. Practically the same results were obtained with other bacteria.

Dr. Richard Reithoffer (*Archiv für Hygiene*, xxvii. H. 4), who quotes the above-mentioned authors, has given the results of new experiments conducted by himself. He tried (1) a common soft soap containing 39 per cent. water and traces of free alcohol; (2) a white almond soap perfumed with nitrobenzol, containing 34.3 per cent. of water and 0.062 per cent. free alkali; and (3) a solid patent potash soap containing 8.4 to 13.8 per cent. water and 0.031 free alkali. The last mentioned, in 1 or 2 per cent. solution, was effective to kill infectious material in bouillon, but 0.2 and 0.5 per cent. did not suffice. Ten per cent. solution killed cholera and other cultures in distilled water in half a minute, and in half and quarter the strength the same result was achieved not so quickly, but in a very short time. All three of the soaps proved to be highly effective in even 1 per cent. solution. Cholera-growths were killed by this strength in from a half to one minute, but the Massaua vibrio required from three to five minutes, whichever of the three soaps was used. In ½ per cent. solution the patent soap killed cholera in five minutes, but the Massaua vibrio at the end of a half-hour was not completely destroyed. The soft soaps proved to be less active, but the almond soap killed all within three to five minutes. From his experiments he concludes that for washing body- and bed-linen, clothing, wooden and iron furniture, so that cholera-germs shall be completely destroyed within five to ten minutes, 4 to 5 per cent. soap solution is probably efficient under all conditions. He points out, however, that one must be careful about soft soaps, the common article of the shops being often unclean and inferior, and suggests the possibility that Kuisl used such in his work.

Further experiments with typhoid bacilli and bacterium coli proved that here, again, the soaps have a high disinfecting-power, and under conditions when other means are not at hand in typhoid they may be used for practical disinfection. But much higher strength must be used than against cholera, and the disinfection of the hands requires very careful washing and plenty of soap. Under favorable conditions it requires 10 per cent. solution of soap to kill typhoid bacilli within a minute. Five per cent. solution requires from three to ten minutes, according to the sort of soap; thus, almond soap three minutes; soft soap, ten minutes. Of the three soaps, the patent potash soap alone seemed to act as strongly against the bacterium coli as against the typhoid bacillus. Unfortunately, the soaps fail utterly, or at least so far as practical disinfection is concerned, against pus cocci. Staphylococcus pyogenes aureus will remain unchanged an hour or more in 18 to 20 per cent. soap solution; hence in surgical practice soaps alone cannot be regarded as efficient.

In the experiments against the bacillus typhosus and bacterium coli the

almond soap proved to be much the most powerful of the three, and the results suggested that nitrobenzol, with which it was perfumed, might have some disinfectant power. Nitrobenzol is almost insoluble in water, but readily soluble in soap solution. Experiment showed that soap solution was made more powerfully disinfectant when ½ per cent. nitrobenzol was added.

Another soap, a "potash cream soap," containing 40 per cent. lysol, was tried against typhoid, bacterium coli, and staphylococci, and proved to be no more powerful than the other soaps excepting against the staphylococci; but here again it proved much weaker than a solution of lysol alone of the same lysol strength. Experiments with carbolic soap showed that carbolic acid also is weakened by the presence of the soap. Thus it appears that the addition of disinfectants to soap is not always advantageous, and that the old way of first washing the hands with soap and then afterward treating with disinfectants is the better one.

A New Process for Producing Sterile Drinking-water.—STABSARZT DR. SCHUMBURG (*Deutsche medicinische Wochenschrift*, March 4, 1897) recommends a new process devised by himself for the quick sterilization of water. It is one which in five minutes kills nearly all water-bacteria and all pathogenic bacteria found in water. The agent used is bromine-water, which, in its turn, after five minutes, is made harmless by means of ammonia, and a clear, tasteless, sterile water is the result. The amount of bromine necessary is very small—only six centigrammes per litre. He uses a solution of twenty parts each of bromine and potassic bromide in one hundred of water, and of this solution 0.20 c.cm. proves to be enough to sterilize a litre of Spree water in five minutes. Very hard waters and grossly polluted river- and marsh-waters require larger amounts, on account of the lime-salts in the former and ammonia in the latter uniting with a part of the bromine before it has an opportunity to exert its disinfectant power. With such waters it is necessary to add the solution until a faint yellow color is produced and persists at least a half minute.

To do away with the bromine of 0.20 c.cm. of the solution an equal volume of 9 per cent. ammonia is enough; and thus, whatever the volume of the bromine solution used, a corresponding volume of 9 per cent. ammonia is necessary.

After treatment the water has practically no taste and is absolutely clear, while the resulting bromine compounds amount to so little as to merit no notice. The method was proved with all sorts of pathogenic organisms, and is recommended particularly for troops in quarters or bivouac, for sterilizing water in the tropics, especially on expeditions, and filling ships' water-tanks, for individual use during the prevalence of epidemics, for the quick preparation of aseptic water for the practising physician, etc. With a kilogramme of bromine (costing in Germany from five to six marks) one can sterilize sixteen thousand litres of water.

Formaldehyd as a Practical Disinfectant.—The fact that the Tollens, Gambier, Trillat, and Krell lamps for generating formaldehyd are not capable of easily producing it in sufficiently large amount for practical use led PROF.

F. C. ROBINSON (*Journal of the American Public Health Association*, October, 1896) to experiment with the view to devise a lamp which might be free from their defects, and thus come into general use for disinfecting-purposes. His lamp consists of a disk of moderately thick asbestos-board perforated with small holes close together and platinized with a strong solution of platinic chloride, and a lamp-font, which is a shallow, cylindrical dish of such size that the disk will just cover the top. This font is partly filled with methyl alcohol The disk is wetted with the alcohol and removed from the dish, and then the alcohol is ignited. By the time the alcohol burns away the disk is sufficiently heated so that when placed over the lamp-font again it will continue hot and change the alcohol to the aldehyd most efficiently. The amount of alcohol converted in a given time depends on the size of the disk; with one of six inches diameter a litre can be converted in an hour.

By way of testing the efficacy of the agent as a disinfectant, Robinson experimented with typhoid- and diphtheria-cultures in a room of three thousand cubic feet capacity, with three large windows with loose sashes. One litre of methyl alcohol was used and the room was closed for four hours. This was found to be insufficient for complete sterilization of infectious material which was covered or concealed to any great extent. But with one and a half litres and three and a half hours' exposure complete sterilization was effected. That the substance has remarkable power of penetration was proved by the fact that all parts of clothing, including seams and inside of pockets, were sterilized in all parts of the room, and typhoid-cultures were destroyed, although covered with a layer of sand a half-inch deep. When two litres of the alcohol were used and exposure continued seven hours, cultures of typhoid and diphtheria placed beneath bedclothes, under the pillows, within the mattress, and even rolled up in a mattress and the whole tied into as compact a bundle as possible, were found to be completely sterilized. From his observations Robinson is of the opinion that at least a quart of alcohol should be used in disinfecting an ordinary living-room; this amount will yield about thirty-six volumes of the aldehyd gas, which, as such, or in solution, is practically without injurious effect on metals, wood, cloth, and most colors.

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All communications should be addressed to

DR. EDWARD P. DAVIS, 250 South 21st Street, Philadelphia, U. S. A.

Or

DR. HECTOR MACKENZIE. 5v Welbeck St., Cavendish Sq., London, W., Eng.

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